CLINICO MYCOLOGICAL STUDY OF

ONYCHOMYCOSIS

Dissertation submitted in

fulfillment of the university regulations for

MD DEGREE IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

(BRANCH XII A)

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CERTIFICATE

Certified that this dissertation entitled **"CLINICO – MYCOLOGICAL STUDY OF ONYCHOMYCOSIS"** is a bonafide work done by **Dr.A.SUDHA**, Post Graduate Student of the Department of Dermatology, Venerology and Leprosy, Stanley Medical College, Chennai – 600 001 during the academic year 2007-2010. This work has not been submitted previously for the award of any degree.

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INTRODUCTION

Onychomycosis is a fungal infection of the nail bed, matrix (or) plate caused by dermatophytes, nondermatophyte moulds and yeasts. [1]

It accounts for 50% of all nail diseases and 30% of mycotic cutaneous infections. [2, 3]

In developing countries, higher priorities to health issues for other diseases have resulted in low awareness of onychomycosis by physician and general public alike. Onychomycosis is all too often regarded as merely a cosmetic problem of relatively minor importance, to seek treatment. This belief may have been supported by the adverse effects and long courses of treatment associated with some earlier antifungal agents. But now safer, effective, short course, systemic treatments are available.

Although onychomycosis is not life threatening, its high incidence and prevalence and associated morbidity makes it an important public health problem, and it is the major cause of nail disease. [4]

Hence an attempt is made to study the prevalence, causative organism and therapeutic response to topical and systemic antifungal agents in onychomycosis.

REVIEW OF LITERATURE

HISTORY

In naming clinical infection due to dermatophytes, tinea precedes the Latin name for the involved body sites. eg. Tinea unguium refers to dermatophytic infection of nail.

The term onychomycosis is derived from the Greek word “Onyx” meaning nail and “mykes” meaning fungus. So it means all fungal infection of nail.

Dermatophyte infections have been described since ancient times. In mid 1800s, David Gruby, along with others Remark Schoenlein, Labert, Malmsten and Robin made major contributions to the field [5].

Sabouraud established the current classification in which dermatophytes are classified by genera. In the 1920s, Hopkins and Benham commenced the actual scientific study of medical mycology. [5]

CLASSIFICATION OF FUNGI

Phylum	**Growth Form**	**Hyphae**	**Sexual Propagules**	**Asexual Propagules**
Zygomycota	Moulds	Broad few septa	Zygospores	Sporangiospores
Ascomycota	Moulds Yeasts	Narrow regular septa	Ascospores	Conidia
Basidiomycota	Moulds Yeasts	Narrow regular septa clamp connections	Basidiospores	Conidia
Deuteromycota	Moulds Yeasts	Narrow regular septa	None	Conidia

EPIDEMIOLOGY

Onychomycosis shows a worldwide prevalence rate of 2 – 18% [6]. In India, incidence varies from 0.5 to 5% in general population[6]. Prevalence rate among children varies from 0 to 2.6%. [7]

Unlike in western countries where it is the frequent cause of nail disorders, in South Asia the prevalence of onychomycosis is relatively low. This was confirmed by large scale survey in Asia in late 1990s in which the prevalence of onychomycosis was lower in tropical countries (3.8%) than in subtropical countries and countries in the temperate zone 18%.[6]

The prevalence rate of onychomycosis is determined by age, occupation, climate, living environment and frequency of travel. [8]

Onychomycosis is more frequent among men than women and increases with age. This is explained by their being relatively more exposed to an environment conducive to the spread of organisms. [9]

Personal and social factors

The reasons for increased incidence in adults are poor peripheral circulation, diabetes, repeated nail trauma, slow nail growth, suboptimal immune function, inactivity, inability to cut the toe nails and to maintain good foot care. [10]

Lower incidence in children is due to reduced exposure to fungus, faster nail growth, smaller nail surface for invasion, lower prevalence of Tinea pedis. [10]

Factors contributing to increased incidence of onychomycosis are,

- Diabetes, peripheral arterial disease, smoking.
- Sports participation - the use of health clubs, commercial swimming pools, and occlusive foot wears for exercise. [11]
- In small percentage of persons, it may be caused by genetic defect that causes an alteration in immune function. [12]
- HIV, Immosupressive therapy, cancer therapy, antibiotics, are other contributory factors.

Source of infection [13]

Anthropophilic species	- largely restricted to human skin.
Zoophilic species	- having animal origins.
Geophilic species	- originating in the soil.

Fungi causing onychomycosis

Up to 90% of mycotic toe nail infections and 50% of finger nail infections are caused by dermatophytes.

Dermatophytes [14]	**Geographical Distribution**
Trichophyton rubrum	World wide
T. mentagrophytes var interdigitale	World wide
T. schoenleinii	Eurasia, North Africa
T. tonsurans	Europe, America
T.violaceum	Africa, Eurasia
Epidermophyton floccosum	World wide

Yeasts

It causes 5% of onychomycosis majority of which is caused by Candida albicans & occasionally in conjunction with mucocutaneous candidiasis. [15]

Nondermatophyte Moulds

4% cases of onychomycosis are caused by nondermatophyte moulds.[16] They are Scytalidium dimidiatum, S.hyalinum[17], Scopulariopsis brevicaulis, Aspergillus, Fusarium[18], Onychocola canadensis[19] Syncephalastrum, Curvalaria, Aureobasidium paullulans[20]

PATHOGENESIS

Invasion of nails by fungus follows a common pattern starting with adhesion between arthoconidia and keratinocytes, followed by penetration through and between cells by a variety of proteolytic enzymes.

Host resistance

- Unsaturated transferrin inhibits the growth of dermatophytes by binding to iron that dermatophytes need for continuous growth. [21]
- Presence of fatty acids from sebaceous glands which inhibit dermatophyte growth is an important mode of defence.

Immunology

- Dermatophytes can activate the alternative pathway of complement activation. [22]
- Produce catalase and superoxide dismutase which may act as defenses against the phagocyte myeloperoxidase system.
- Development of cellular immunity via sensitised T lymphocytes is a key factor in immunological defence. [23]
- Langerhan's cells can act as antigen presenting cells for dermatophyte antigens. [24]

CLINICAL PRESENTATION

It presents as whitish or yellowish streak ,a patch of discolouration at free edge near lateral nail fold, spreads towards base and becomes brown, black. The nail plate becomes thickened, lifted up by subungual hyperkeratosis, gross invasion may lead to massive destruction of nail plate.[25] Characteristically some nails are always spared.

Tinea unguium also associates with dermatophytic infection in other areas of the body like Tinea pedis, Tinea manum etc... Candidal nail infection may be associated as paronychia & intertrigo.

CLINICAL TYPES[25]

Onychomycosis has been divided into five clinical types.

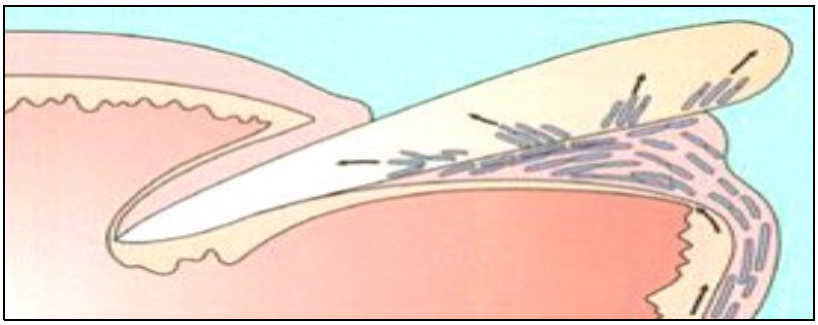
1) Distal and lateral subungual onychomycosis (DLSO).
2) Proximal subungual onychomycosis (PSO).
3) Superficial white onychomycosis (SWO).
4) Total dystrophic onychomycosis (TDO).
5) Endonyx onychomycosis.

DISTAL AND LATERAL SUBUNGUAL ONYCHOMYCOSIS

This is the most common type, accounting for about 90% of cases of tinea unguium. The most common etiological agent is Trichophyton rubrum.

It starts by invasion of stratum corneum of the hyponychium of the distal nail bed (or) the lateral nail fold. Subsequently the infection moves proximally in the nail bed and invades the ventral surface of the nail plate, manifests as whitish to brownish yellow opanification.

Subungual hyperkeratosis results from a hyperproliferative reaction of the nail bed in response to the infection and leads to onycholysis. Splinter hemorrhage may be seen probably due to mild inflammation compressing vessels. As the infective process continues, the invasion of the nail plate results in gross and total destruction of the nail. Increasing invasion of the ventral nail plate makes it thick, discoloured and friable. The subungual debris also provides a site for secondary infection by bacteria, other moulds and yeasts.



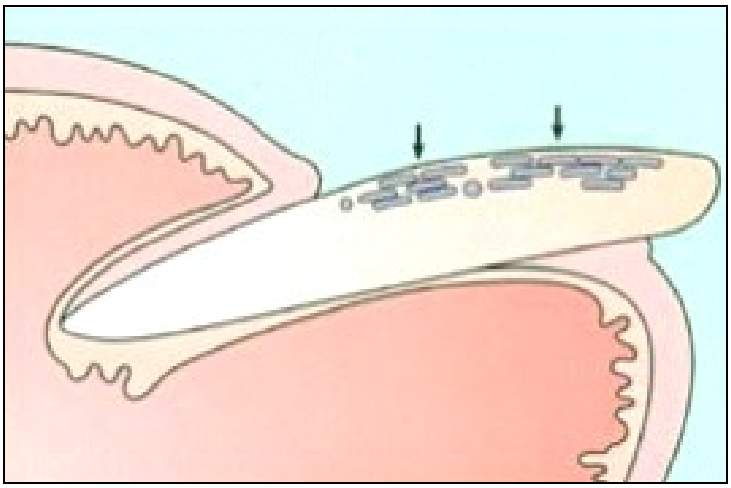
Nail invasion in distal lateral subungual onychomycosis

SUPERFICIAL WHITE ONYCHOMYCOSIS

It is second most common type and accounts for 10% of all cases [26]. It produces distinct form of nail invasion in which the dorsal surface of the nail plate is eroded in well circumscribed powdery white patches often away from the free edge. The whole surface of the nail plate may be thus affected.

Although more common with Trichophyton mentagraphytes var interdigitale, it can occasionally be seen in Trichophyton rubrum infections and also occurs with certain nondermatophytes.

Toe nails are usually affected but in AIDS patients superficial white onychomycosis of both finger and toe nails has been reported. In AIDS patients superficial infection may coexist with proximal nail plate invasion. Nail becomes rough and crumbly.

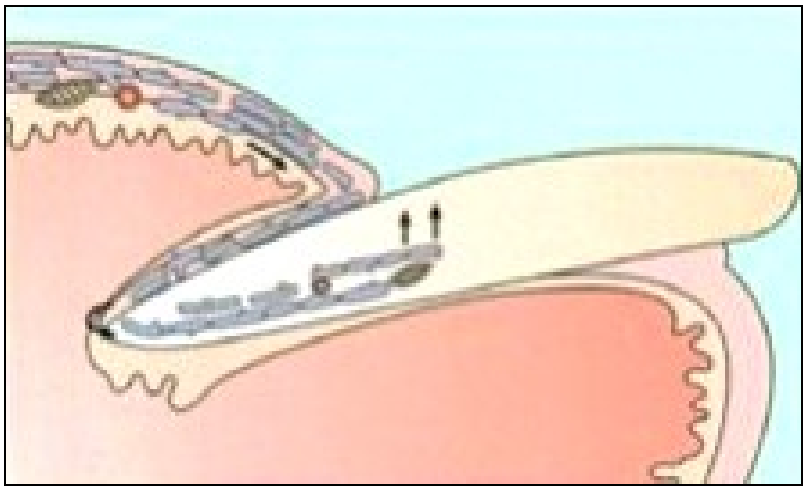


Nail invasion in superficial white onychomycosis

PROXIMAL SUBUNGUAL ONYCHOMYCOSIS

This pattern is very uncommon particularly associated with AIDS patients. [26] It is most commonly caused by Trichophyton rubrum, [26] and also by T.mentagrophytes, T.tonsurans.

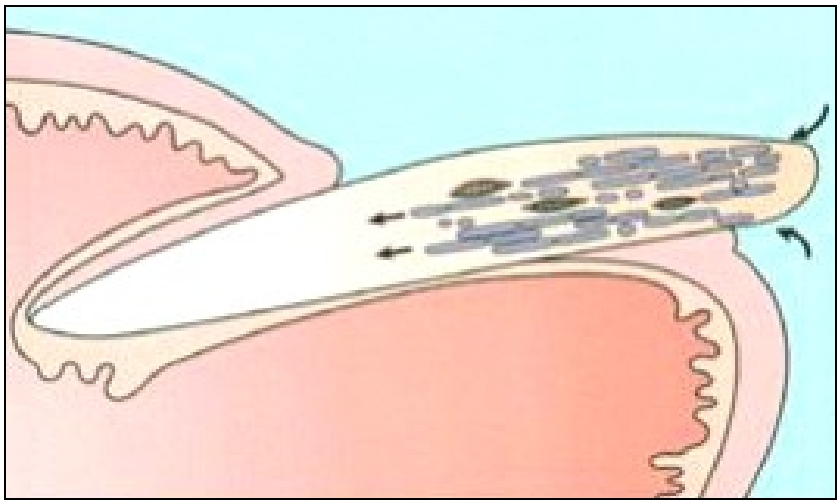
The first clinical sign is a whitish to brownish area on the proximal part of the nail plate. This area gradually enlarges to affect the entire nail. It is also associated with peripheral vascular compromise



Nail invasion in proximal subungual onychomycosis

ENDONYX ONYCHOMYCOSIS

This is seen with infection caused by dermatophytes that cause endothrix scalp infection notably Trichophyton soundanense[27]. The nail plate is scarred with pits and lamellar splits. The invasion occurs from the top surface but penetrates deeply into the nail plate.



Schematic drawing of nail invasion in endonyx onychomycosis

TOTAL DYSTROPHIC ONYCHOMYCOSIS

It is characterized by total destruction of the nail plate which usually may be the end result of other patterns of onychomycosis. The entire nail unit becomes thick and dystrophic.

ONYCHOMYCOSIS IN DIABETIC PATIENTS

Approximately one third with diabetes are affected with onychomycosis. Diabetic patients suffering from decreased foot sensation are more prone to trauma, which damage the nail and nail matrix, opening portals of entry for the fungus to infect the nail. The risk factors are peripheral neuropathy,impaired peripheral circulation, family history, intake of immunosuppressive drugs, and duration of diabetes.[28]

Male diabetics have three times higher risk than female diabetics. Diabetes with onychomycosis have 15% rate of secondary infection compared with 6% in diabetics without onychomycosis and three times greater risk of gangrene and foot ulcer.

ONYCHOMYCOSIS IN HIV

It is characterized by being clinically more aggressive with a higher frequency of unusual presentations and resistance to conventional treatment. Multiple fungal species and unusual opportunistic fungi are frequently cultured from HIV infected patients.

The prevalence of onychomycosis in HIV patients has been reported to be 15-40%. It can be an early manifestation of immunosuppression and is more frequent when CD_4 cell count approaches 450 cells/µ.[29]

ONYCHOMYCOSIS IN CHILDHOOD

Superficial white onychomycosis is more common and it rarely involves multiple digits. A family history of onychomycosis may be present and may be a factor in transmission as well as susceptibility.

Severe onychomycosis in children raises concern with HIV or other forms of immunosuppression. The prevalence of childhood onychomycosis is 0 to 2.6% and tends to be associated with concomitant Tinea pedis or Tinea capitis. [7]

COMPLICATIONS [30]

- Nail disfigurement.
- Nail loss
- Secondary bacterial infections.
- Cellulitis
- Chronic utricaria
- Can spread to other areas of the body.

INVESTIGATIONS

Collection of Nail Specimen [31]

Distal and lateral subungual onychomycosis

Samples should be obtained from the nail bed and ventral nail plate. It is very important to try to collect material from the most proximal portion of the affected nail bed. This is the area most likely to contain viable fungi. The affected nail bed is exposed by removing the overlying onycolytic nail plate with the nail clipper, and then appropriate material is taken by scrapping the hyperkeratotic nail bed with the solid or disposable scalpel no.15 or small curettage.

Superficial white onychomycosis

Shallow shaving with the disposable scalpel or gently scrapping the dorsum of nail with the curette will provide specimens for microscopy and culture.

Proximal subungual onychomycosis

The affected nail plate is exposed by perforating the proximal nail with the disposable punch or an electric drill. Samples are then obtained by scrapping the exposed nail plate with the disposable scalpel.

Endonyx onychomycosis

Viable fungi are present throughout the whole thickness of the nail plate. Nail clippings can therefore be sufficient . In candidal onychomycosis infected material should be collected from the proximal edge.

DIRECT EXAMINATION

The material obtained is placed on a glass slide containing 40% KOH solution and is covered by a cover slip. The alkaline solution will digest the protein, lipids and most of the other epithelial debris that are present but the fungal filaments resist this treatment as they have chitinous wall.

The slide may be gently heated taking care to avoid boiling which leads to crystallization. Nail clippings take a long time to dissolve. When the material has been softened by KOH, pressure on the cover slip is applied to squash the specimen and then it is examined under the low power microscope.

Counter stains like chlorazol black and parker's blue enhance the visibility of the fungus. Calcofluor fluorescent stain enhances the fungal cell wall by staining chitin[32]. Microscopy can differentiate between dermatophytes (hyaline hyphae) nondermatophytes (long sinus hyphae) and yeast (budding cells with pseudo hyphae) but species identification cannot be made from wet mounts.

CULTURE

Samples were inoculated into media – sabouraud's dextrose agar with chloramphenicol and cycloheximide, SDA with chloramphenicol without cycloheximide both were inoculated at 25° C and 37°C and examined for dermatophytes,candida and moulds. If no growth observed after 6 weeks, the sample was reported as negative. In most cases the culture was repeated more than once and in cases where contamination was suspected subculture was done. [33]

Bacterial growth is inhibited by the addition of chloramphenicol and gentamycin to the media. Yeast and nondermatophytic moulds tend to grow more rapidly than dermatophytes in a matter of days versus weeks respectively. [37]

CULTURAL CHARACTERISTICS OF COMMONLY ISOLATED PATHOGENS

Dermatophytes [35]

Trichophyton rubrum

a) Downy form

Colony

Surface of the colony is white, downy or cottony. The reverse of the colony is initially dark brown with pale cream border, but after incubation for 3-4 weeks produces the typical deep red pigment characteristic of this species.

Microscopy

Small tear shaped, clavate or elongate microconidia are arranged along the sides of the hyphae.

b) **Melanoid form**

Colony

Brown melanoid pigment masks red pigment on reverse of the colony.

Microscopy

Small tear shaped microconidia are arranged along the sides of the hyphae.

c) Dysgonic form

Colony

Slow growing tiny deep red colonies with a brittle texture. This form is relatively unstable and will quickly revert to the more typical downy form.

d) Granular form

Colony

The surface is powdery or granular cream to pink and often raised and folded in the centre. The reverse is red brown.

Microscopy

Numerous smooth thin walled cylindrical or pencil shaped macroconidia are produced.

e) Yellow form

Colony

Smooth, leathery and yellow. Reverse is yellow.

Trichophyton mentagrophytes [36]

Colony

Rapidly growing with white powdery and creamy centre. The reverse is reddish brown, often with a pale edge.

Microscopy

Spherical microconidia arranged in bunches, spiral hyphae and macroconidia are present in some isolates. The fluffier downy isolates have pyriform microconidia arranged along the sides of the hyphae.

Trichophyton schoenleinii

Colony

Glaborous or velvety usually heaped and folded with a fringe of hyphae at the edge. The surface is white to cream and reverse pale.

Microscopy

Conidia are usually absent. Characteristic dichotomously branching hyphae with flattened tips, termed chandelier or antler hyphae are present.

Trichophyton violaceum

Colony

Slow growing glaborous colonies have a waxy or leathery texture. They are deep red in color.

Microscopy

Microconidia and macroconidia are usually absent. Chalmydoconidia and disorted hyphae may be present.

Trichophyton verrucosum [37]

Colony

Very slow growing fungus, white or grey waxy colonies.

Microscopy

Short hyphae with terminal chalmydoconidia. Clavate or elongate micro conidia may be present along the sides of the hyphae.

Rat tailed macroconidia may be produced on depleted media.

Non dermatophyte moulds

Scopulariopsis brevicaulis

Colony

On cycloheximide free medium, the fungus grows rapidly to produce a powdery cinnamon brown surface, often with radical or cerebriform folds. The reverse is cream to brown.

Microscopy

Chains of basipetal conidia are formed from annellides. The conidia are rough, lemon shaped with truncate base.

Fusarium

Requires cycloheximide free medium. In one week pale pink or brownish flat colonies develop.

Microscopy

Numerous sickle shaped macroconidia and elliptical or oval micro conidia.

These arise from short phialidic cells in colonies of fusarium oxysporum, long phialidic cells in colonies of fusarium solani.

Acremonium

Grow in media both with and without cycloheximide. In cycloheximide free medium white pink velvety colonies develop within one week.

Microscopy

Reveal elliptical conidia grouped at the tip of long phialides.

Scytalidium

In cycloheximide free medium fast growing colonies with an abundant aerial mycelium develop.

S.dimidatum

Initially white and become black and dark brown in a few days.

S.hyalinum

Remain white or creamy in colour.

Microscopy

Reveal the chains of arthrospores.

Aspergillus terreus

Colony

Rapidly growing powdery with a cinnamon brown surface and pale yellow reverse.

Microscopy

The vesicle at the apex of the stout conidiophore bears metulae and phialides only on the upper two third of its surface. Long chains of smooth brown phialoconidia are produced which form a columnar head.

Aspergillus niger

Colony

Coarse black granules against the creamy colony surface.

Microscopy

Globose vesicles with biseriate phialides large echinulate jet black conidia in chains. Foot cells are present.

Rhizopus

Colony

Cottony to wooly gray colonies that rapidly fill the Petri dish or test tube. Salt and pepper colony surface.

Microscopy

Broad irregular hyphae that are aseptate or sparsely septate. Well developed rhizoids. Branching sporangiophores with hemispherical columellae arising form nodes adjacent to rhizoids.

Curvalaria

Colony

Species are velvety or woolly with dark brown to black to olive green pigment.

Microscopy

Dark hyphae and slender geniculate conidiophores.

Syncephalastrum

Colony

Yellow to yellow green velvety to powdery colonies and form concentric rings of growth where conidiation is heavy.

Microscopy

Unicellular hyaline phialoconidia on short, plump flask shaped phialophores. Clusters of subglobose to ellipitical conidia in balls at the tip of the phialides.

Aureobasidium pullulans

Colony

White to yellow colonies with a wrinkled or folded topography. As arthroconidia are produced, colonies become shiny, mucoid, dark brown to black.

Microscopy

Delicate thin walled hyphae and ellipitical conidia.

Candida albicans

Colony

White to cream and soft in texture.

Microscopy

Reveals budding yeast cells. The production of filaments is best examined on depleted media such as cornmeal agar or rice extract agar supplemented with Tween 80.

It differs from most other species of candida by production of rudimentary true hyphae (germ tubes) when inoculated into serum and incubated at 37^{o}C for 2 – 4 hrs.

HISTOPATHOLOGY

Histological examination of the nail is useful alternative to culture / KOH. [41]

Nail clippings may be sent to laboratory for diagnosis in formalin filled container (or) an incisional biopsy may be performed to confirm the diagnosis.

Staining in the laboratory should be performed with periodic acid shiff, methanamine silver, Haematoxyllin eosin, toludine blue stain to reveal fungal elements [34].Presence of glycogen and mucoproteins in the cell walls of fungal hyphae appears red indicating a positive stain. [28]

Haemotoxylin eosin shows basophilic, refractile structures. If fungi are present in the horny layer they usually are sandwiched between two zones of cornified cells, the upper being orthokeratotic and the lower consisting partially of para keratotic cells called “Sandwich sign”.

Histopathologic examination shows psoriasiform hyperplasia, parakeratosis, thinned ridges, narrow suprapapillary plates and dilated tortuous capillaries. It dosen’t identify the species of causative pathogen.

Unna was the first to give a detailed histopathological description of infected nails.

Advantages

- The result is much faster than culture usually 3 days versus 3 weeks.
- Histopathology is more often positive than culture.
- Only means to differentiate onychomycosis from nail psoriasis, lichen planus, alopecia areata, nail eczema and other inflammatory nail disorders.

Immuno histochemistry [34]

With this procedure, nail sample sections are exposed to labeled antibodies specific to suspected fungal pathogens. Monoclonal anti Trichophyton spp (dermatophytes), anticandida spp (yeasts), anti aspergillus spp (moulds) antibodies are used for the identification of the fungi. Visualization is achieved by direct immunofluorescence, immunoperoxidase or avidin biotin complex methods.

Flow cytometry

Flow cytometry is based on the ability to identify molecular variances among fungal species. Dissociated nail samples are filtered to collect single fungal cells that are analyzed with a flow cell sorter on the basis of DNA and protein detection, cell size and cell granulosity.

In vivo confocal microscopy

It is a highly discriminatory technique that facilitates the examination of living tissue. Reflected light is used to penetrate the nail at various depths, thus imaging layers of the nail optically. Detection of fungal hyphae in the nail plate and nail bed depends on light penetration into the tissue and the reflectivity of the structures being observed.

Scanning electron microscopy

It generates three dimensional images of high resolution, high magnification and large focal depth. Cross sectioned nail specimens are fixed, dehydrated and dried for viewing with a scanning electron microscope, allowing for detailed imaging of the spatial orientation, penetration and integration of fungal hyphal elements in the nail plate.

Polymerase chain reaction methods

PCR provides sensitive and specific detection of DNA sequences. DNA extracted from nail specimens is amplified by PCR methods, detection & differentiation of dermatophytes, nondermatophyte moulds and yeasts is achieved by amplification with specific fungal DNA primer sets.Subsequent digestion by restriction enzymes in restriction fragment length polymorphism analysis allows identification of fungal species, strain and sub type.

DIAGNOSIS OF ONYCHOMYCOSIS

Based on criteria formed by Scher et al (J Am Acad Dermatol June 2007)

Clinical

Primary Criteria

- White / yellow or orange/brown patches or streaks.

Secondary criteria

- Onycholysis
- Subungual hyperkeratosis / debris
- Nail plate thickening

Laboratory

- Positive microscopic evidence
- Positive culture of fungus

Nondermatophyte moulds have been cultured from onychomycotic nails, but at times may be contaminants. Certain criteria need to be fulfilled before these are considered as primary pathogens. [38]

The criterias are, [39]

- If a dermatophyte is isolated on culture from nail clippings, it is considered a pathogen.

- ❖ In case of culture of a mould or yeast, it should be considered a pathogen only if hyphae, spores or yeast cells are seen on microscopic examinations.
- ❖ Confirmation of an infection by a nondermatophyte mould requires isolation of the organism on at least 5 out of 20 inocula without concurrent isolation of a dermatophyte.
- ❖ Isolation of same mould from three consecutive nail samples.

As a rule, nondermatophytic onychomycosis favours diseased or aged nails, toe nails are the usual sites of involvement.

Most isolates can be identified from the primary culture, but if sporulation is poor, a number of media may be used to encourage the production of conidia, including potato dextrose agar and lactrimel agar.[40]

DIFFERENTIAL DIAGNOSIS [43]

a) **Hand Eczemas**

May develop dystrophic nails with transverse ridges. Presence of or history of eczematous lesions would help in differentiation.

b) Psoriasis

Presence of symmetrical nail involvement and skin lesions in psoriasis help in differentiation. Pits are characteristic and distinguishing features. Pits are uncommon in fungal infections.

c) Lichen Planus

Thinning is the commonest change and there are usually associated cutaneous lesions, other findings include longitudinal ridging, pterygium unguis, nail loss, twenty nail dystrophy, hyperkeratosis, onycholysis.

(d) Leukonychia (Acquired / congenital)

Superficial white onychomycosis needs to be distinguished.

(e) Paronychia

May be caused by bacterial infection.

(f) **Darier's disease**

Nails show 'V' shaped wedges.

(h) Pachyonychia congenita

(i) Reiter's syndrome

(j) Norwegian scabies

(k) Pseudomonas infection

(l) Neoplastic

Subungual melanoma, Subungual fibroma

(m) Yellow nail syndrome

(n) Usage, Degenerative, Age related disorder

(o) Onycholysis

(p) **Atherosclerosis, venous disorder, peripheral vascular disease.**

(q) Repeated trauma.

(r) Allergic contact dermatitis, collagen vascular diseases.

MANAGEMENT OF ONYCHOMYCOSIS

There are several factors that need to be considered in the management of onychomycosis.

a) Drugs (efficacy, adverse effects profile, dosage schedule, duration of therapy, relapse rates, monitoring schedule and cost effectiveness).

b) Patient and disease profile (the location and extent of onychomycosis, etiologic agents, any concomitant health problems, and concurrent medications.

TOPICAL ANTIFUNGAL AGENTS

The have sometimes been considered for the treatment of early onychomycosis, in the absence of nail matrix involvement. The more commonly used topical agents for the treatment of onychomycosis are 5% amorolfine nail lacquer, 8% ciclopiroxolamine nail lacquer, 28% ticonazole and bifonazole with 40% urea paste. [44]

AMOROLFINE

Amorolfine is a morpholine derivative. It is active against dermatophytes and dimorphic fungi with activity against yeasts and moulds being more variable. [45]

Mechanism of action

Amorolfine interferes with the steps in the sterol biosynthesis pathway. These include the enzymes Delta 14 reductase and Delta 7 – 8 isomerase. An alteration in the sterol content of the fungal membrane may result in hyper fluidity and permeability changes with the fungus eventually becoming nonviable.[47]

When amorolfine is applied to the nail surface the solvent evaporates to leave a highly concentrated deposit of drug in an occlusive film on the nail. This acts as deposit from which amorolfine penetrates and diffuses through the nail plate to nail bed over the next seven days. [26]

Duration

Once or twice weekly 6 months for finger nails, 12 months for toe nails.

Indications

For mild cases of distal and lateral subungual onychomycosis, > 18 years of age.

Contraindication

Hypersensitivity, pregnancy, breast feeding, under 18 years of age.

Adverse effects

Slight redness, transient burning sensation in the areas of the nails after application of the lacquer. [47]

Interaction

Not to use nail polish and artificial nails during treatment. It is not systemically absorbed and no known interaction with other drugs. [26]

CICLOPIROXOLAMINE

It is a hydroxypyridone topical anti fungal and fungicidal against Trichophyton rubrum, T.mentagrophytes, Epidermophyton floccosum, Micosporum canis and Candida albicans.

Mechanism of action

It inhibits transport of certain essential substrates into fungal cells and interferes with the synthesis of DNA, RNA and proteins in growing cells. The main mode of action is its high affinity for Fe^{3+}, Al^{3+} [48]. The inhibition of these essential cofactors affects mitochondrial electron transport processes thereby impairing microbial metabolism. [49]

It also reduces the activity of catalase and peroxidase which are responsible for the intracellular degradation of toxic peroxides.

Adverse effects

Periungual erythema, burning or tingling sensation at the application site. [50]

TICONAZOLE (28%) SOLUTION

It is an imidazole with the broad spectrum of antifungal activity. The organisms that responded includes Trichophyton rubrum, Scytalidium dimidiatum and acremonium species.

Other topical therapies are 1% bifonazole and 40% urea, clotrimazole cream and solution, miconazole tincture, glutaraldehyde 10% solution, 1% fluorouracil in propylene glycol ,a tincture containing triacetin, sodium propionate, benzalkonium chloride, cetylpyridinium chloride and chlorozylenol, vitamin E, naftifine hydrochloride 1% gel, topical ketoconazole under occlusion following nail avulsion.

SYSTEMIC ANTIFUNGAL AGENTS

GRISEOFULVIN

It is an oral antifungal agent derived from penicillium griseofulvum and has narrow spectrum of action against dermatophytes. [51]

Mechanism of action

It inhibits the formation of intracellular microtubules, disrupts the mitotic spindle and prevents cell division of the fungus. [52]

Pharmacokinetics

Griseofulvin is poorly absorbed after oral administration because of its poor solubility in water. The bioavailability of this agent is improved by taking with a fatty meal and also intake in micronized form.

The peak serum concentration occurs approximately 4 hours after drug administration. 84% is bound to plasma protein, albumin and has low affinity for keratin. Prolonged administration of griseofulvin is required because the drug is fungistatic and persists in the nail for a very short time approximately 2 weeks after discontinuation of treatment. [52]

Griseofulvin is predominantly metabolized in liver and excreted in urine. Its half life is 9 to 22hrs.

Indications

Griseofulvin has a narrow spectrum with effectiveness extending to dermatophytes only. [54]

Interactions

Contraindicated in individuals with porphyria, hepatocellular failure and hypersensitivity.

Warfarin needs dosage adjustment. Dose of grisefulvin is to be increased while on barbiturates. It should not be administered during pregnancy because of the risk for developing conjoint twins. [55]

Adverse effects

Hypersensitivity, oral thrush, nausea vomiting, epigastric distress, diarrhoea, headache, dizziness, insomnia, mental confusion. [55]

Dose: 500mg / day 6 months for fingernail onychomycosis, 12 months for toe nail onychomycosis.

FLUCONAZOLE

It inhibits fungal lanosterol 14 alpha demethylase. The oral bioavailability is greater than 90%, not seem to be affected by gastric PH. The peak plasma concentration is between 1 and 2 hours, half-life is between 20 and 50 hours.

Fluconazole has low lipophilicity and low level of plasma protein binding, primarily cleared by the renal system.

Indications

Fluconazole has proved safe and effective in the treatment of fingernail and toe nail onychomycosis. [56]

Dose

150 mg, 300 mg (or) 450 mg once weekly for 9 – 12 months.

Interaction

Contraindicated with cisapride, terfenadine. Cemiditine, rifampicin decreases plasma concentration of fluconazole. Hydrochlorothiazide increases plasma concentration of fluconazole. It increases plasma concentration of cyclosporin, astemizole, glipizide, glyburide, phenytoin, rifabutin, tacrolimus, theophylline, zidovudine.

Prothrombin time is increased with anticoagulant.

Adverse effects

Headache, skin rash, nausea, vomiting, diarrhoea, palpitations, sweating, fever.

ITRACONAZOLE

It is a triazole derivative & disrupts the synthesis of ergosterol.

It is highly lipophilic, oral bioavailability is maximal when taken with a full meal. [57] Approximately 95% is bound to plasma albumin. It has a high affinity for keratinized tissues and has been detected in distal finger nail material after 1 week of therapy. It persists for upto 6 months after discontinuation of therapy more or less unchanged. [58]

Itraconazole is predominantly metabolised by the CYP $3A_4$ iso-enzyme, the major metabolite is hydroxyitraconazole. 18% is excreted through feces. Approximately 40% of the dose is excreted as inactive metabolites in the urine.

Indications

Effective for dermatophytes, candida, nondermatophyte molds may also respond well to itraconazole.

Drug Interactions

Quinidine, pimozide, midazolam, triazolam, cisapride, astemizole, terfenadine, lovastatin, simvastatin are contraindicated .

Plasma concentration is decreased by carbamazepine, phenobarbitone, pheytoin, rifabutin, rifampin, antacids, H_2 blockers, proton pump inhibitors, nevirapine.

Plasma concentration is increased by erythromycin, clarithromycin, indinavir, ritonavir. Oral contraceptives may be ineffective.

Adverse effects

Headache, rhinitis, sinusitis, diarrhoea, flatulence, abdominal pain, dizziness, rash, cystitis, urinary tract infection, gastritis, fever, pain, tremor, herpes zoster, abnormal dreaming. Rare cases of congestive heart failure and pulmonary edema have been reported. [57]

TERBINAFINE

It is an allylamine class of antifungal agent discovered in 1970s.

Terbinafine is primarily fungicidal against dermatophytes, aspergillus species, scopulariopsis brevicaulis, blastomyces, histoplasma capsulatum. The activity against yeast is more variable.

The primary target of terbinafine in fungi is the membrane bound enzyme, squalene epoxidase. This produces ergosterol deficiency, which interferes with membrane function. On the other hand, accumulation of squalene results in the deposition in lipid vesicles with disruption of cellular membranes. [59]

Terbinafine is well absorbed (>70%) and the bioavailability is 40% as a result of first pass metabolism. Greater than 99% of terbinafine is nonspecifically bound to plasma proteins. Peak plasma concentration is within 2hrs after a single dose. The allylamine is extensively distributed and extremely lipophilic.

Terbinafine is extensively metabolized, its main metabolite is demethyl terbinafine. Approximately 70% of the administrated dose is eliminated in the urine.

When terbinafine 250mg / day was administered, the drug was detected in peripheral nail clippings after 7 days of medications. Mean half life is 22 days. [60]

Half of the drug concentration is still retained for 90 days at levels higher than the MIC values of most nail pathogens by a factor of 15 – 50 times

Dosage

250mg/day for 6 weeks and 12 weeks in fingernail and toenail onychomycosis respectively.

Interactions

Its plasma concentration is decreased by rifampicin and Increased by cimetidine, terfenadine. It increases the concentration of caffeine, theophylline and decreases the concentration of cyclosporin.

Adverse effects

Nauses, diarrhoea, abdominal pain, dyspepsia, rash, uticaria, eczema.

Rare cases of symptomatic hepatic injury have been reported. Terbinafine may also cause headache, taste disturbances and white cell disturbances.

Adverse effects associated with terbinafine are usually mild, transient and reversible on discontinuation of therapy. [61]

TREATMENT OF ONYCHOMYCOSIS IN CHILDREN[7]

Topical antifungals can better penetrate the thin nail plate of children and are used as first choice treatment.

Terbinafine, Itraconacole and Fluconazole have all been used safely in children.

ADJUVANT THERAPY

Surgical or chemical nail avulsion with 40% urea may be useful in patients with severe onycholysis, extensive nail thickening or longitudinal streaks in the nail.

Topical antifungal agents are of limited efficacy when used alone but may result in a more rapid cure when used in conjunction with the newer systemic compounds.

STRATEGIES TO IMPROVE EFFICACY OF TREATMENT [62]

a) Accurately identify pathogenic organism to ensure proper selection of antifungal agent.
b) To distinguish from psoriasis or other diseases.
c) Recognize nail characteristics
    - Thick nails
    - Lateral infection

- Dermatophytoma
- Severe Onycholysis

d) Recognize patient characteristics
   - Poor perfusion
   - Diabetic
   - Elderly
   - Immunocompromised

e) Recognize and implement techniques to increase success:
   - Debride thick nails, onycolytic nails, dermatophytomas.
   - Choose medications that are safe and convenient for the patient and appropriate for the infecting organism.
   - Assess the possibility or need for prophylactic therapy (topical for long term follow up of cured nails, treatment for tinea pedis).
   - Combination therapies (If standard therapy fails).

f) Educate patient about onychomycosis:
   - Need for compliance with therapy and follow up.
   - Educate about toe nail hygiene to prevent recurrence.

Poor prognostic factors [42]

- Area of nail involvement > 50%
- Significant lateral disease.
- Subungual hyperkeratosis >2mm
- White/yellow or orange/ brown streaks in the nail.
- Total dystrophic type.
- Non responsive organisms.
- Patients with immunosuppression.
- Diminished peripheral circulation.

Recurrence

It is not uncommon. Recurrence often implies that although the clinical signs have resolved, either mycological cure was not achieved with the initial treatment or a new infection has developed during or immediately after treatment period.

Mycological cure

Define as negative KOH microscopy and culture results.

Clinical cure

Expressed as % of nail plate that is visibly clear of infection.

Complete cure

Includes mycological and clinical cure.

AIMS AND OBJECTIVES

To Study

- Age and sex distribution of the patients with onychomycosis
- Clinical types of onychomycosis
- Isolation of causative organisms
- Precipitating factors
- Associated dermatological conditions
- Associated systemic diseases.
- Therapeutic trial with systemic terbinafine alone, topical amorolfine alone and both together.

MATERIALS AND METHODS

This randomized single-blind longitudinal clinical comparative study was undertaken during the period of Aug 2008 to July 2009. 108 patients presenting to our department out patient clinic with clinical features of finger nail or toe nail onychomycosis (eg. Discolouration, thickening, crumbling or destruction of nail plate, subungual debris and onycholysis) were subjected to detailed history, clinical examination and investigations like potassium hydroxide (KOH) mount and culture from nail clippings/ scrappings.

89 patients were either KOH, culture or both positive. Among these who had received systemic/ topical antifungal therapy in the last six months, pregnant, lactating, those with elevated hepatic enzymes, not willing for study were excluded from the drug trial. Only 60 patients were randomly selected for study and divided into three groups (A, B, C). Group A received oral terbinafine alone 250mg daily for 6/12 wks, Group B received topical amorolfine alone once weekly for 6 months, Group C received oral terbinafine daily for 6/12wks plus topical amorolfine once weekly for 6 months.

The patients were evaluated at 6weeks, 12 weeks and 24 weeks. During these visits they were assessed for the growth of the normal and healthy nail plate and were inquired for any adverse effects of

the drugs. In addition, microscopic examination and culture of nail material were done at 12, 24 weeks.

Direct microscopy was done after overnight incubation of the nail specimen in 40% KOH for the presence of fungal mycelia and spores. All the nail specimens were cultured on sabouraud dextrose agar (SDA) with chloramphenicol and with or without cycloheximide. The cultures were observed twice a week for a period of 4 weeks and were discarded if there was no growth at the end of 4 weeks. The culture tube was examined for colour of the colony (on the surface and reverse), texture, and rate of growth.

In the presence of growth, a loopful of growth was taken and examined using a lactophenol cotton blue mount. Slide culture was also done if required and was examined for characteristic morphology.

The criteria used for the diagnosis of dermatophytes were,

- If a dermatophyte was identified on KOH mount and/or isolated on culture, it was pathogenic.

The criteria used for the diagnosis of Nondermatophytic moulds [NDM] were,

- demonstration of fungal filaments on KOH mount and
- Isolation of NDM in culture.

Isolation of NDM on three occasions was considered as pathogenic.

Proposed definitions of cure when assessing patients with onychomycosis in clinical trials (**Based on criteria formed by Scher et al (J Am Acad Dermatol June 2007)**

Criteria for cure

A) 100% absence of clinical signs of onychomycosis (mycology not required) OR

B) Negative mycological laboratory results with one or more of following clinical signs

i) Distal subungual hyperkeratosis or onycholysis leaving less than 10% of nail plate affected

ii) Nail plate thickening that does not improve with treatment because of comorbid condition

Criteria for noncure

A) Presence of positive mycological results.

B) Any one of the 4 clinical signs, even in the presence of negative mycological results

i) Residual major changes (>10%) of the nail plate compatible with dermatophyte infection

ii) White / yellow or orange brown / patches or streaks in or beneath the nail.

iii) Lateral onycholysis with debris in an otherwise clear nail plate.

iv) Hyperkeratosis on the lateral nail plate / nailfold edge.

The results were recorded and a detailed analysis was done.

OBSERVATIONS AND RESULTS

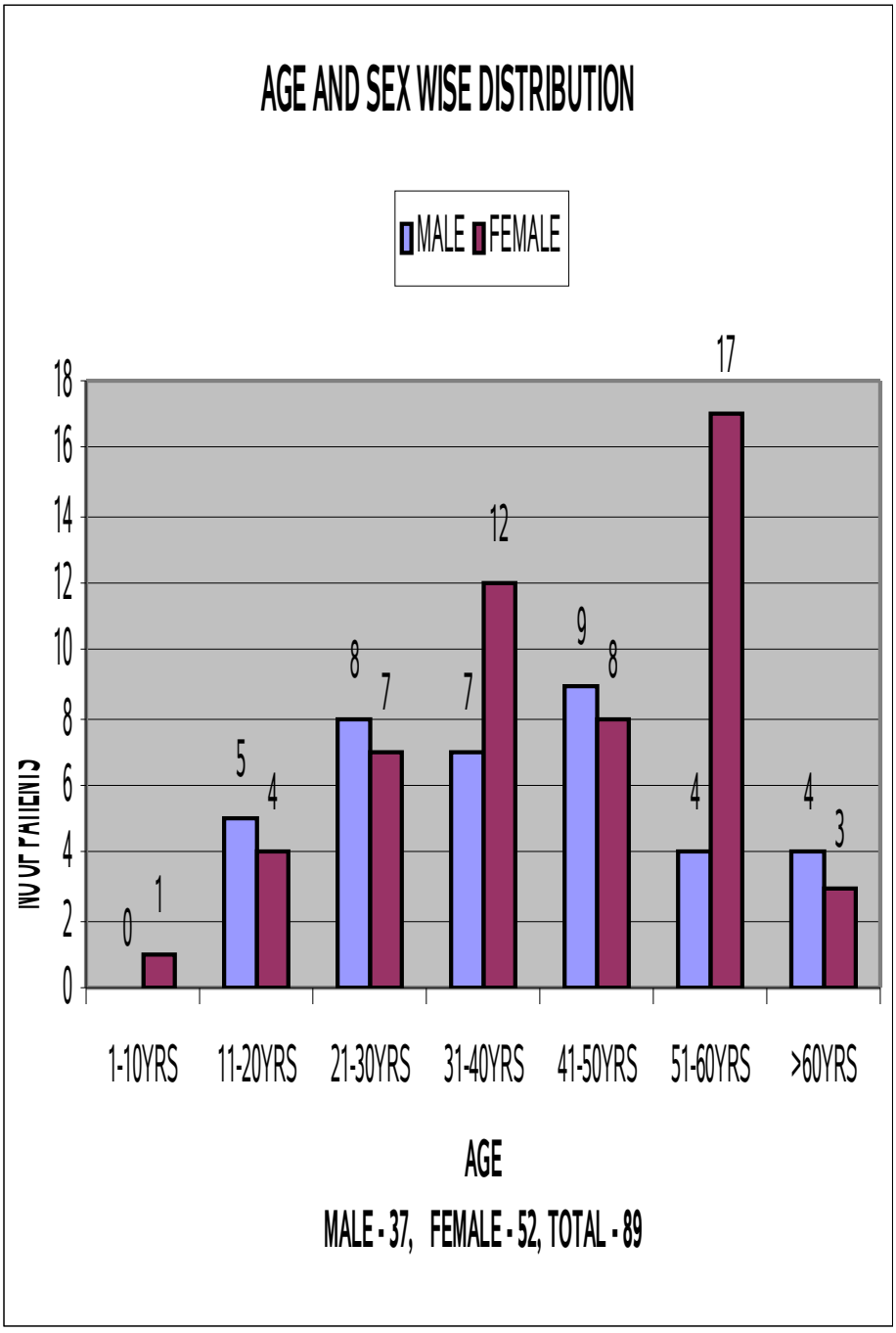
Out of 89 patients, 37 were male patients (41.5%) & 52 were female patients (58.5%) giving a sex ratio of 1:1.4.

Table 1

AGE & SEX WISE DISTRIBUTION

Age in years	No. of Male	No. of Female
0-10	-	1
11-20 yrs	5	4
21-30 yrs	8	7
31-40yrs	7	12
41-50yrs	9	8
51-60yrs	4	17
>60	4	3
Total	37	52

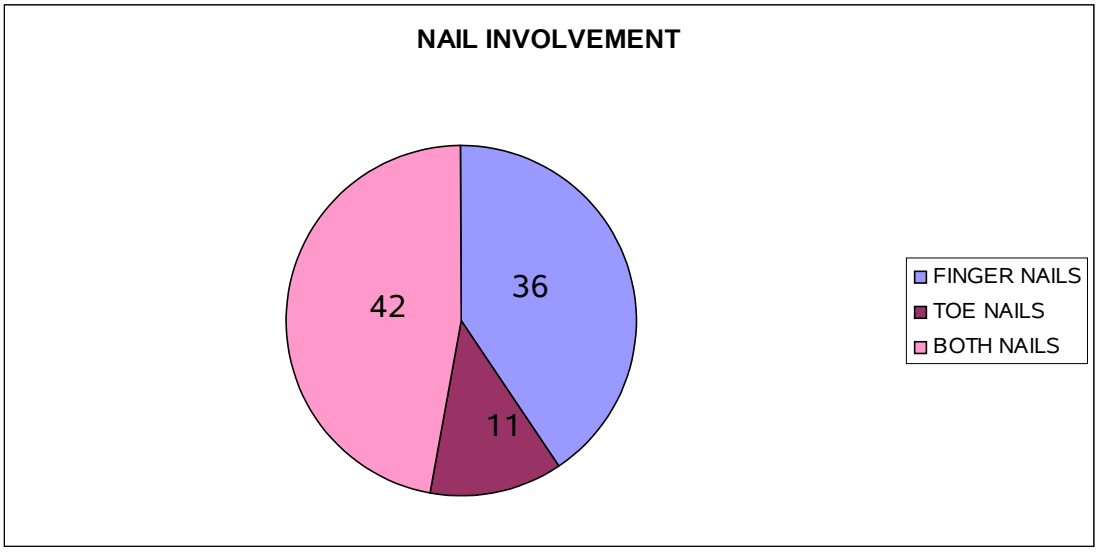
The most commonly infected was the 51-60 yrs age group[21 patients] followed by 31-40 yrs age group[19 patients] 41-50 yrs (17 patients), 21-30 yrs (15 patients), <20yrs (10 patients) and above 60 yrs (7 patients). Infection was less common in the age group below 10 years, whereas infection was high above 50 years. Youngest age in this study was 3 years. Oldest age was 70.


AGE AND SEX WISE DISTRIBUTION
MALE
FEMALE
18
16
14
12
10
8
6
4
2
0
0
1
5
4
8
7
7
12
9
8
4
17
4
3
1-10YRS
11-20YRS
21-30YRS
31-40YRS
41-50YRS
51-60YRS
>60YRS
AGE
MALE - 37, FEMALE - 52, TOTAL - 89

With regard to occupational exposure, 37 patients (41.5%) were housewives, 12 patients (13.5%) were hotel staffs.Manual labourers (8.9%), and farmers (5.6%) were next commonly affected.

The duration of illness was < 6 months in 41 patients (46%), 6 months to 1 year in 21 Patients (23.6%),1-2 years in 7 patients(7.9%), >2 years in 20 patients (22.5%).

12 patients gave history of trauma prior to nail infection and family history of onychomycosis was reported by 4 patients. Smoking history was found in 12 patients, alcohol intake in 9 patients.



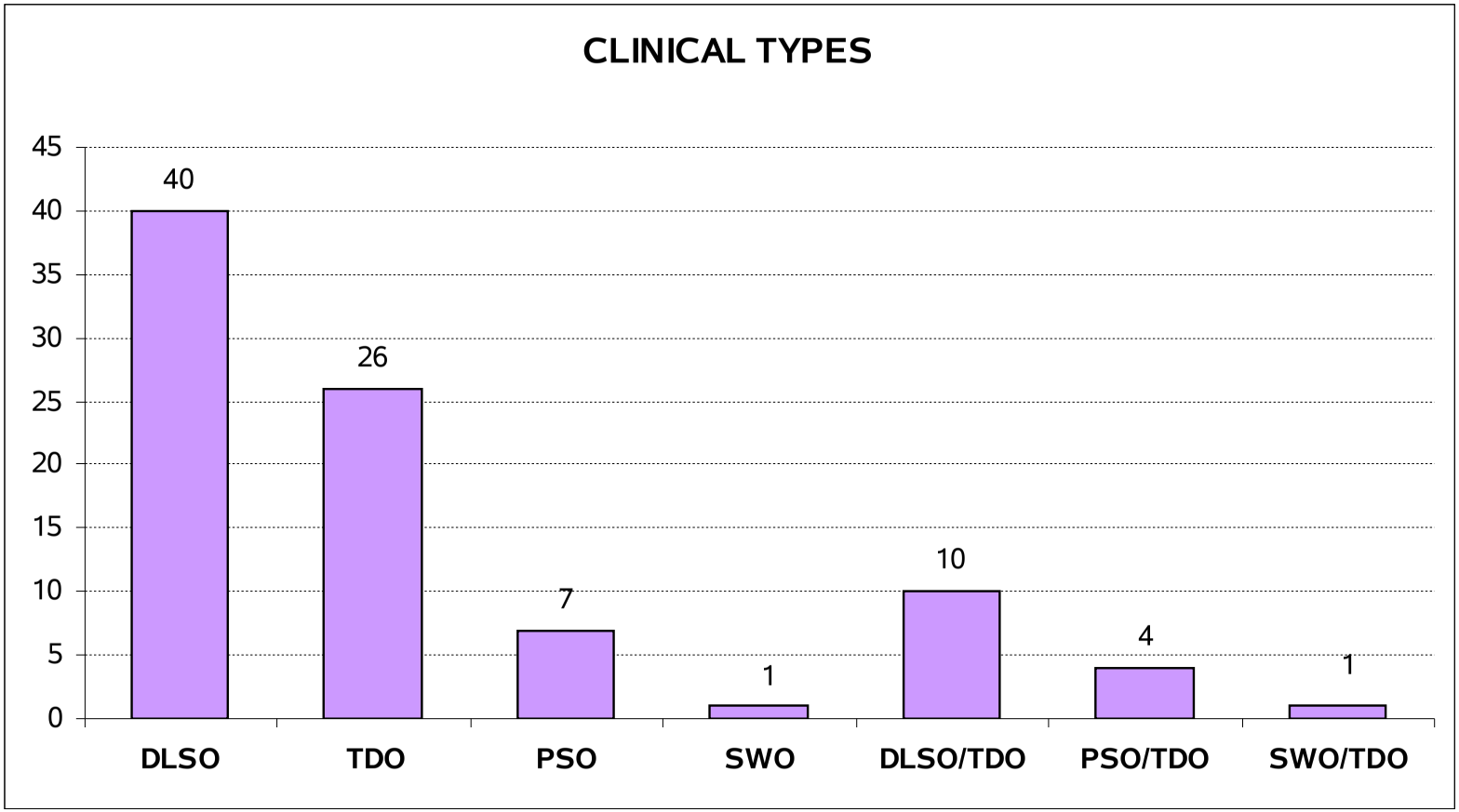
36 patients showed only fingernail involvement, 11 patients only toe nail involvement, 42 patients had both fingernail and toe nail involvement at the time of presentation.

TABLE - 2

CLINICAL TYPES

Type	No of Patients	Percentage
DLSO	40	44.9%
TDO	26	29.2%
PSO	7	7.8%
SWO	1	1.1%
DLSO & TDO	10	11.2%
PSO / TDO	4	4.5%
SWO / TDO	1	1.1%

Distal and lateral subungual onychomycosis (DLSO) was the most frequent clinical pattern noted in 40 patients followed by Total dystrophic onychomycosis (TDO) in 26 patients, DLSO&TDO in 10 patients, proximal subungual type in 7 patients. Least common type was superficial white onychomycosis noted in 1 patient.



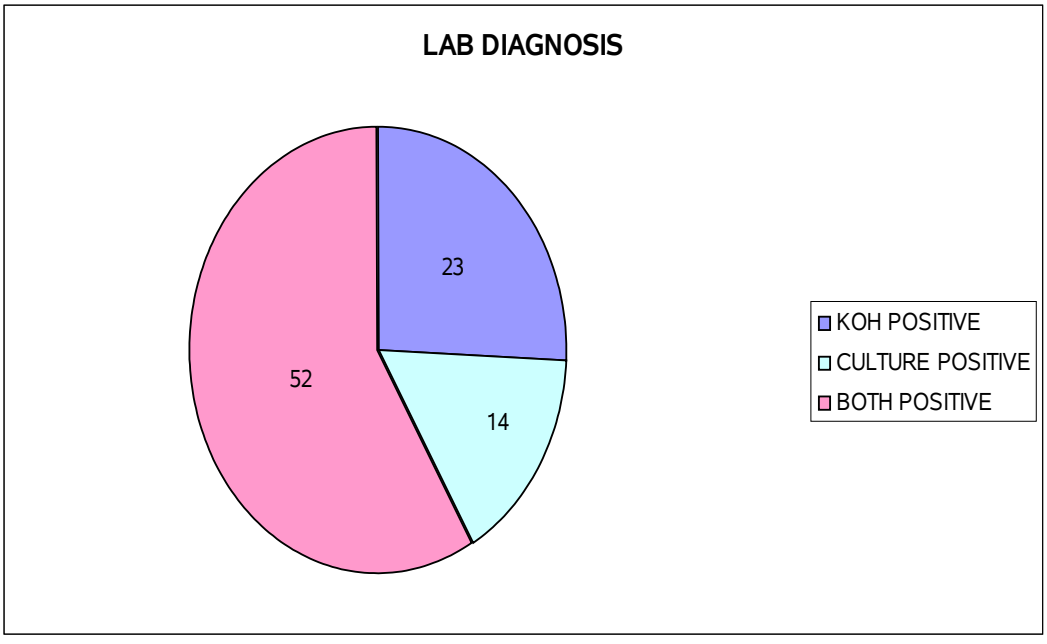
DLSO – Distal and lateral subungual onychomycosis
TDO – Total dystrophic onychomycosis
PSO – Proximal subungual onychomycosis
SWO – Superficial white onychomycosis

Dermatological conditions associated with onychomycosis

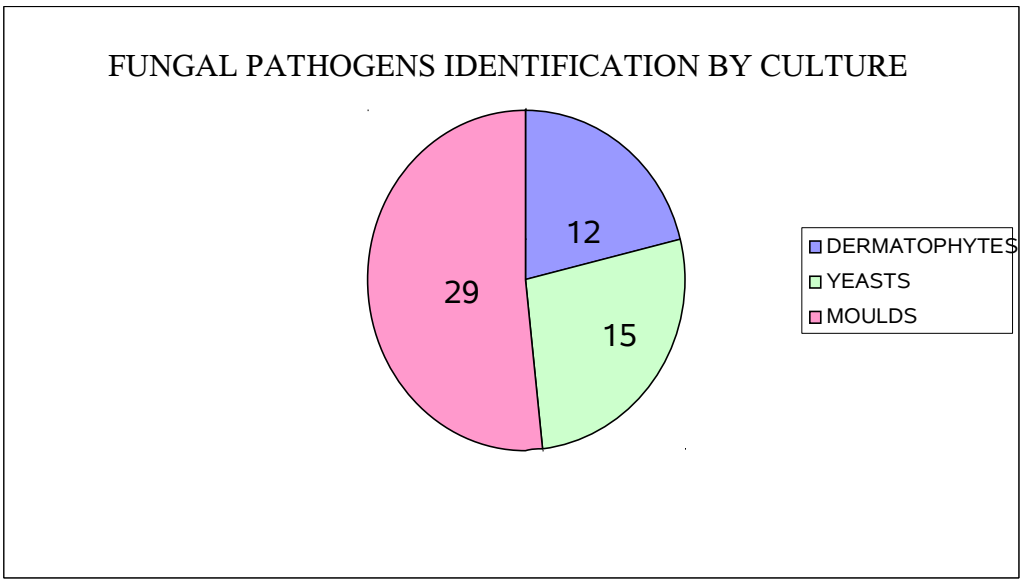
S.No	Dermatological condition	No. of Patients
1	Vitiligo	3
2	Psoriasis	3
3	Hansen's disease	2
4	Intertrigo	2
5	Keratolysis punctata	2
6	Perforating folliculitis	1
7	Morphea	1
8	Nevus achromicus	1
9	Tinea versicolar	2

Systemic diseases associated with onychomycosis

S.No	Associated systemic disorders	No. of Patients
1	Diabetes mellitus	11
2	Hypertension	10
3	Renal transplant	2
4	Hypothyroidism	1
5	Seizure disorder	1
6	Filaria	1



Of the 89 specimens studied fungal elements were identified by positive KOH mounts with culture negative in 23 specimens and 14 specimens showed KOH negative and culture positivity. Both culture & KOH positivity was seen in 52 specimens.



Yeasts & dermatophytes were isolated in 15, 12 specimens respectively, while nondermatophytic moulds were isolated in 29 samples.

ISOLATED FUNGAL PATHOGENS

1. Yeasts

Organism	No of patients	Percentage
Candida	7	46.6%
Nonalbicans candida	8	53.4%

2. Moulds

organisms	No of patients	percentage
Aspergillus niger	15	51.7%
Rhizopus	5	17.2%
Syncephalastrum	4	13.8%
Curvalaria	2	6.9%
Aureobasidium pullulans	2	6.9%
Scopulariopsis	1	3.4%

3. Dermatophytes

Organisms	No of patients	Percentage
Trichophyton rubrum	5	41.6%
T.verrucosum	2	16.6%
T.schoenlenii	2	16.6%
T.mentogrophytes	2	16.6%
T.violaceum	1	8.3%

During the course of the study, two patients in Group A, four patients in Group B, one patient in Group C were lost in follow up period & were excluded from the analysis of results.

At sixth month follow up,

Mycological response in the study group

Fungal Isolate	Group A N=18		Group B N=16		Group c N=19	
	Cured	Total	Cured	Total	Cured	Total
Dermatophytes	4	4	3	5	3	3
Yeasts	3	4	2	4	4	5
Nondermatophytes (moulds)	5	9	4	7	6	8
KOH positive	1	1	-	-	3	3
Overall response	13	18	9	16	16	19
Overall percentage	72%		56%		84%	

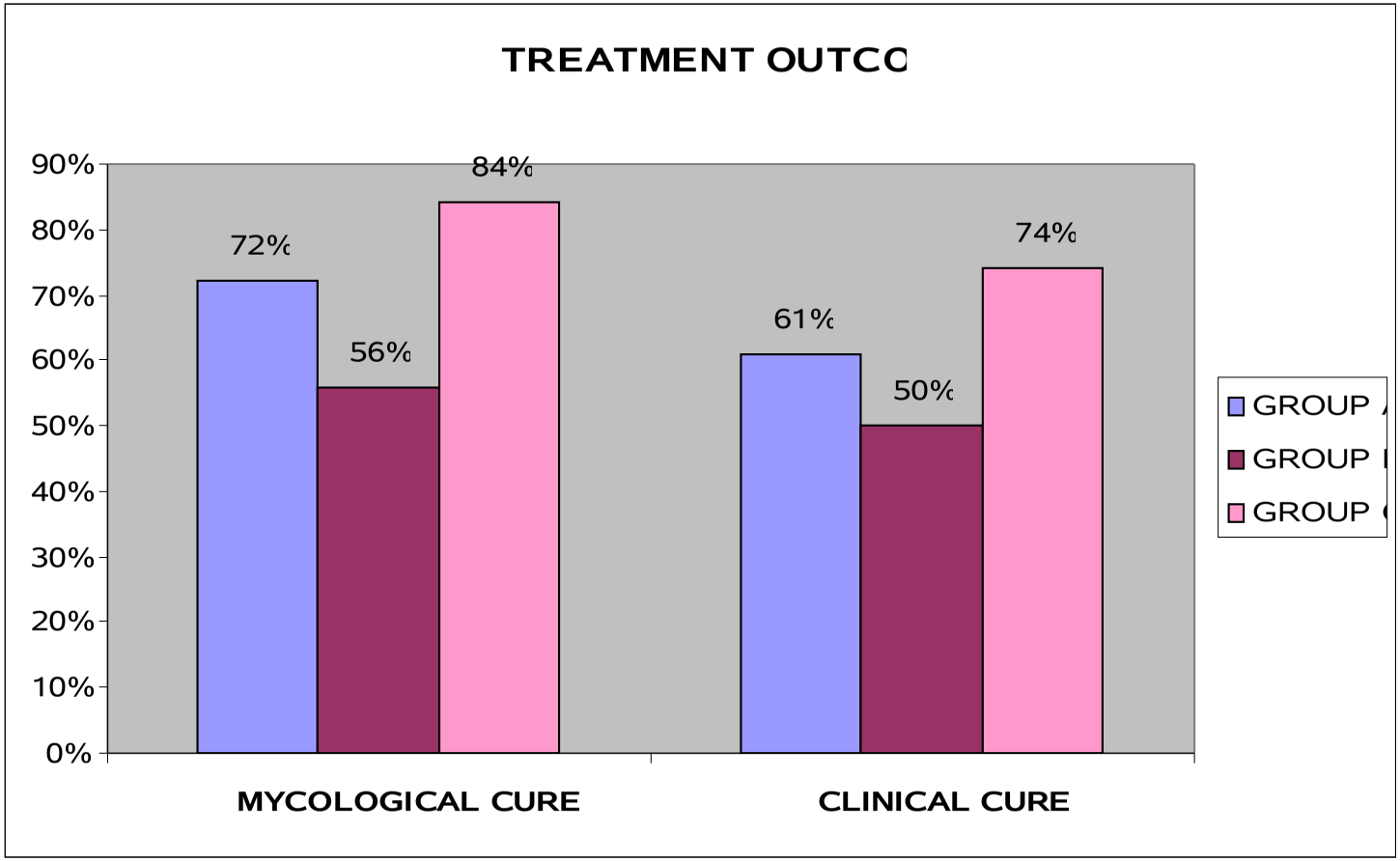
Mycological cure rate was 72%, 56%, 84% in Group A, B, C respectively.

Clinical response in the study group

Group	Cured	Total	Percentage
A	11	18	61%
B	8	16	50%
C	14	19	74%

Clinical cure was observed in 61%, 50%, 74% patients in group A, B, C respectively.

No major adverse effect due to drug was observed in this study.



Group A – Oral terbinafine alone
Group B – Topical amorolfine alone
Group C – Both oral terbinafine and topical amorolfine

Distal and lateral subungual onychomycosis.

Superficial white onychomycosis.

Proximal subungual onychomycosis.

Total dystrophic subungual onychomycosis.

Tinea unguium with Tinea manum

3 yrs old child with dystrophic thumb & middle finger and paronychia.

GROUP –A

Dystrophic thumb nail before treatment.

Normalization of proximal edge of nail after 6 weeks of treatment with oral terbinafine

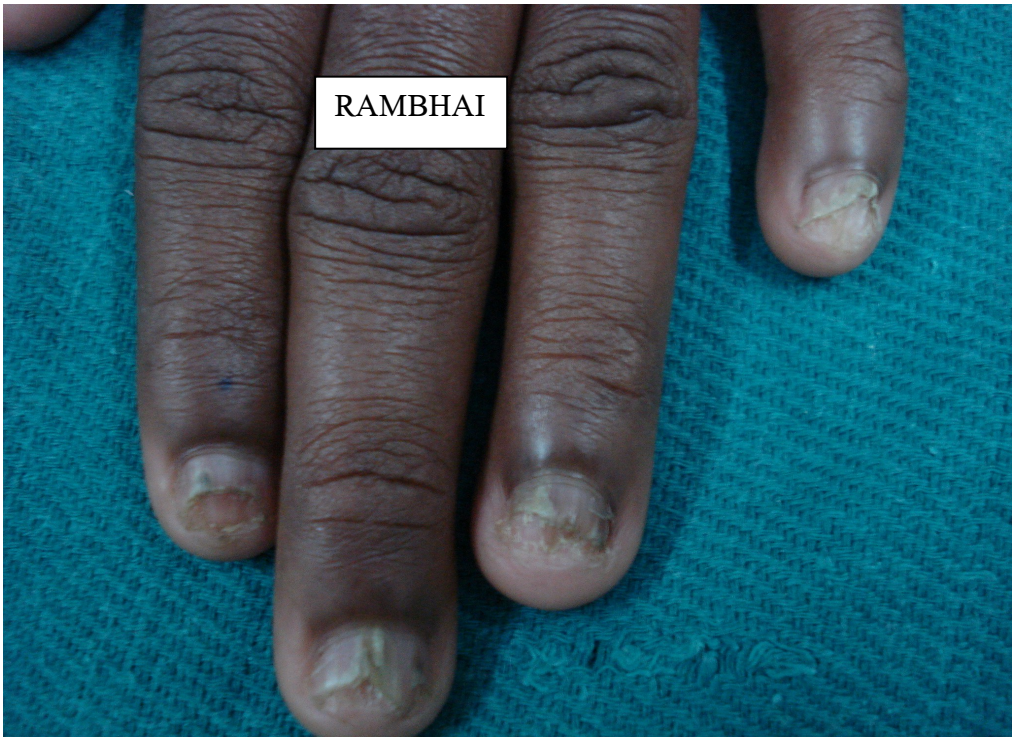
GROUP –B

Blackish discolouration and thickened finger nails.

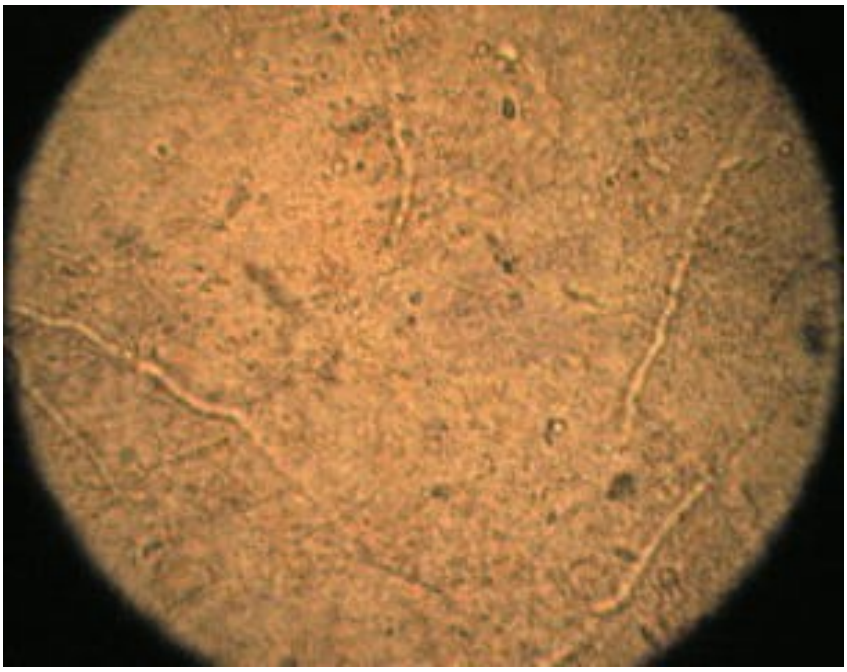
Change in discolouration & decrease in subungual hyperkeratosis noted after 6 months of topical amorlfine application.

GROUP –C

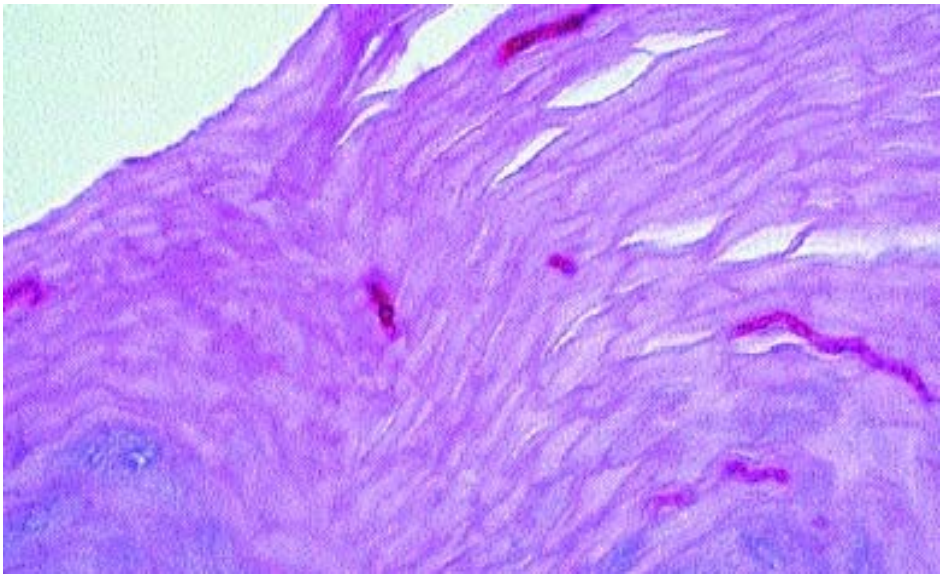
Onycholysed finger nails before treatment



Normalization of nail plate after treatment with oral terbinafine and topical amorolfine for 6 weeks



REFRACTILE, SEPTATE FUNGAL HYPHAE SEEN IN POTASSIUM HYDROXIDE MOUNT.



HYPHAE SEEN WITHIN STRATUM CORNEUM IN PERIODIC ACID SCHIFF STAIN

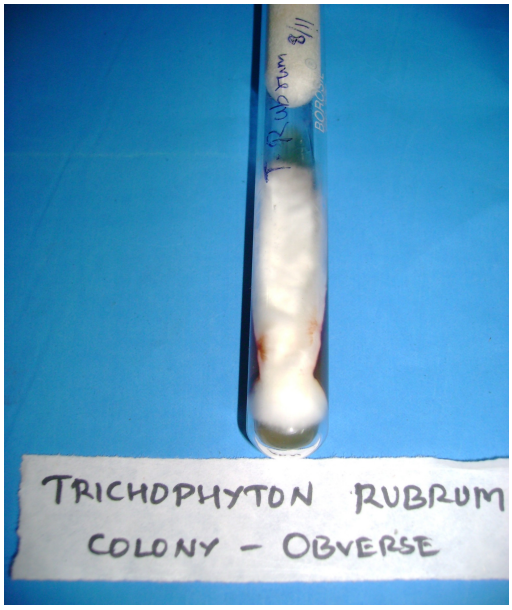
CULTURE
(MACROSCOPY)

Candida albicans

WHITE TO CREAM
COLOURED COLONY

Syncephalastrum

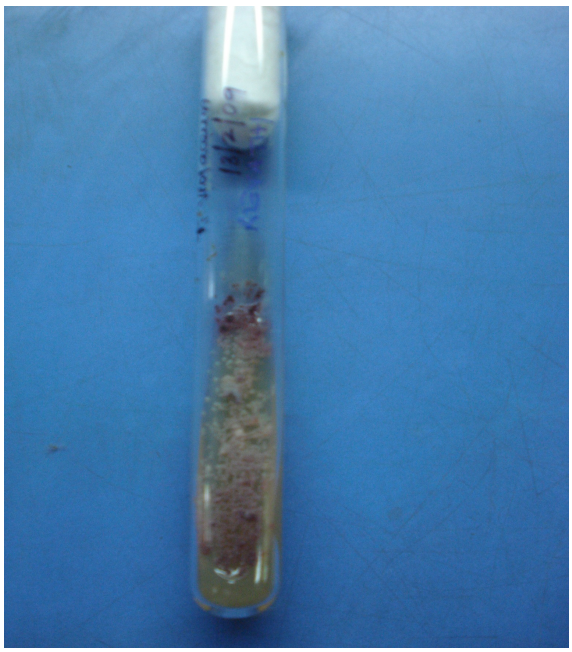
YELLOW GREEN VELVETY
POWDERY COLONY



WHITE COTTONY COLONY

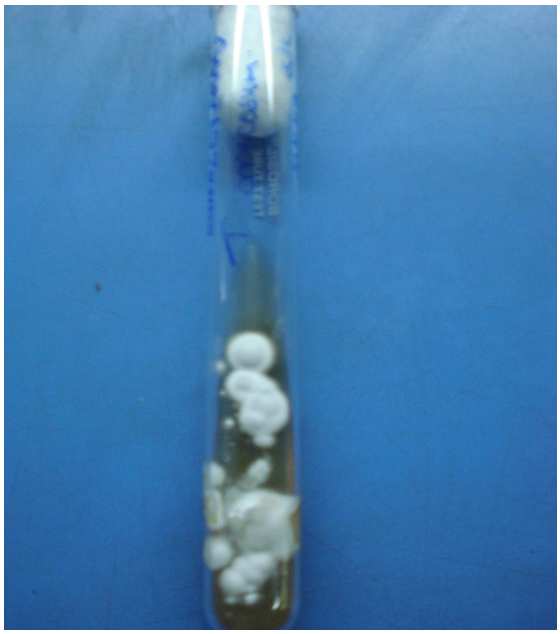
DEEP RED PIGMENT

Trichophyton violaceum



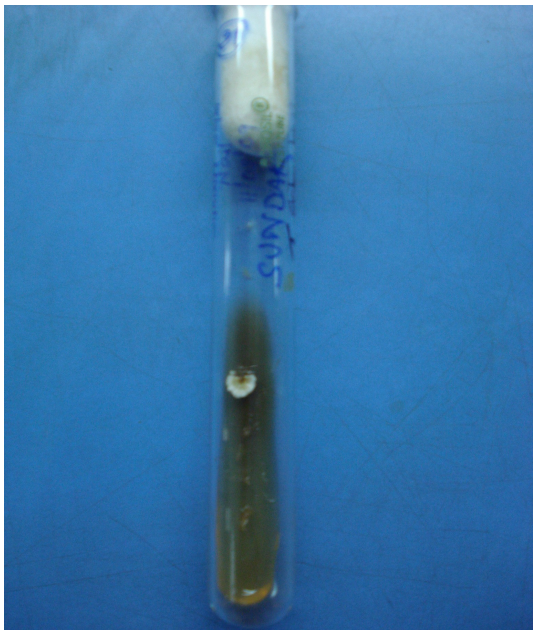
WAXY LEATHERY COLONY

DEEP RED COLOUR ON REVERSE



Trichophyton verrucosum

WHITE WAXY COLONY

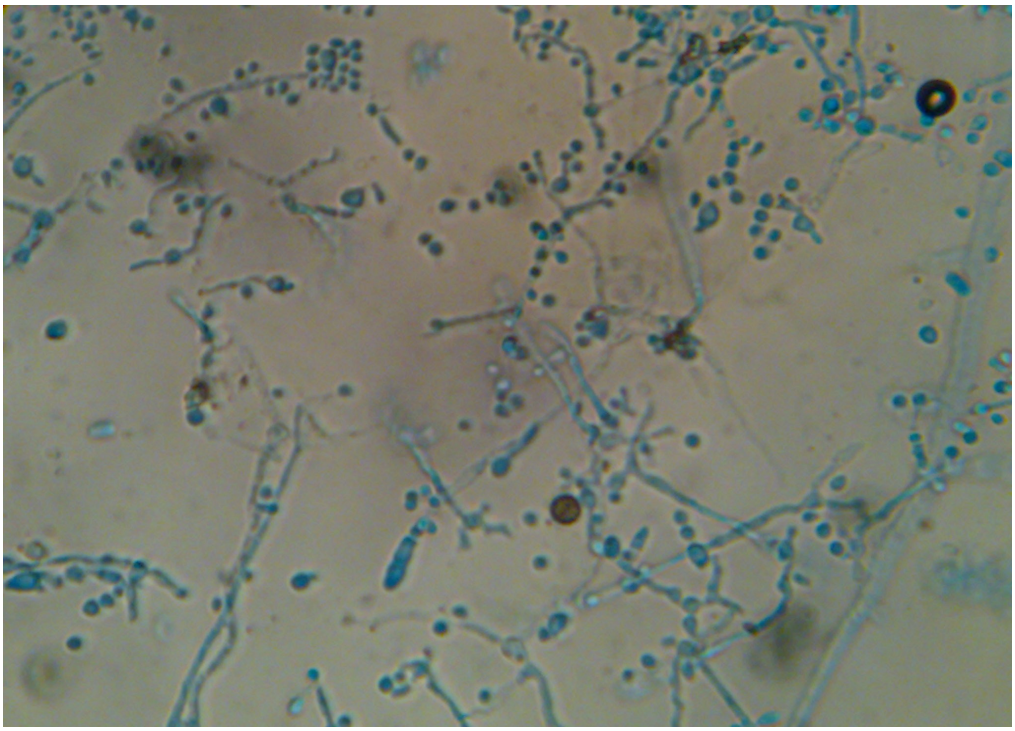


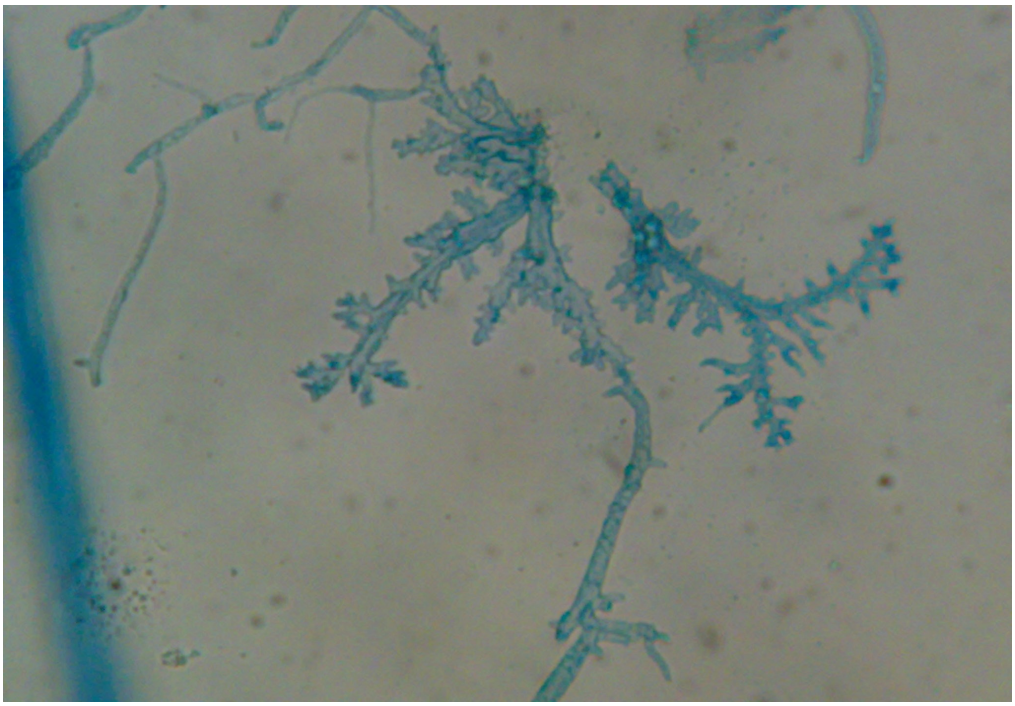
Trichophyton schoenleinii

WHITE VELVETY HEAPED AND FOLDED COLONY

(Trichophyton rubrum)

SMALL TEAR SHAPED, CLAVATE MICROCONIDIA ALONG THE SIDES OF THE HYPHAE ON LACTOPHENOL COTTON BLUE MOUNT



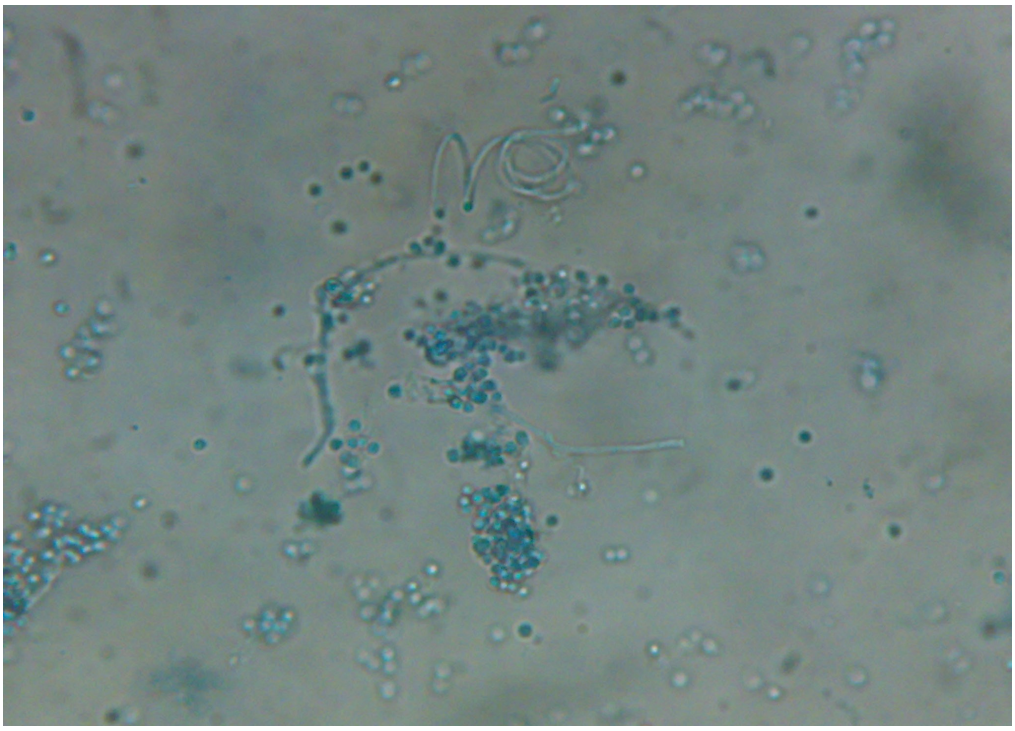


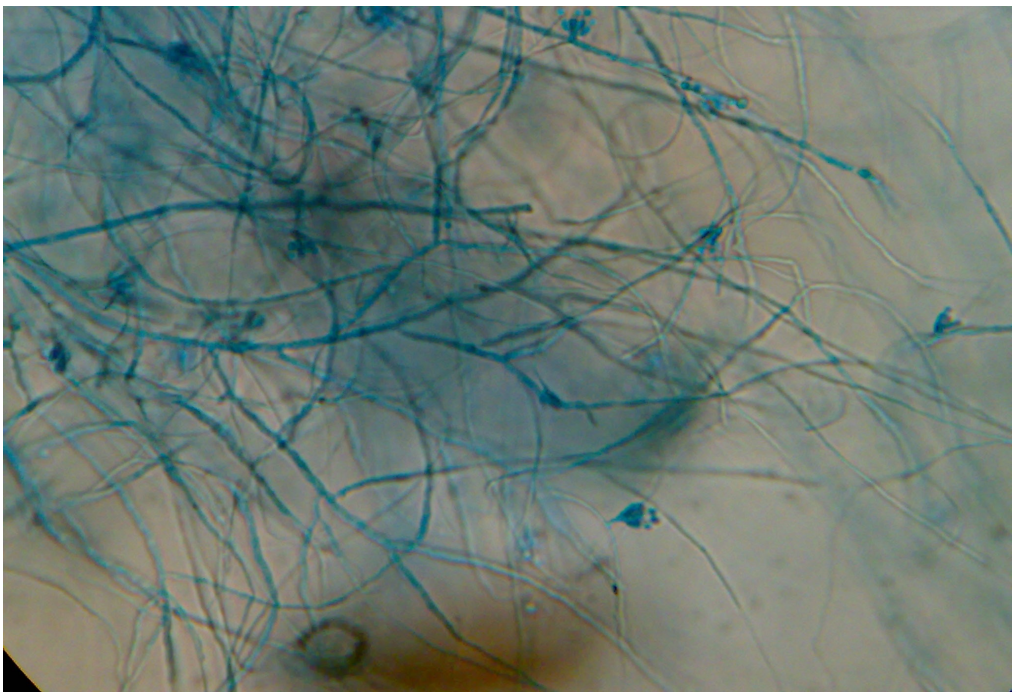
(Trichophyton verrucosum)

SHORT HYPHAE WITH TERMINAL CHALMYDOCONIDIA ON LACTOPHENOL COTTON BLUE MOUNT

(Trichophyton mentagrophytes)

SPHERICAL MICROCONIDIA, SPIRAL HYPHAE SEEN ON
LACTOPHENOL COTTON BLUE MOUNT





Trichophyton schoenlenii

CHANDELIAR (OR) ANTLER HYPHAE SEEN

DISCUSSION

Onychomycosis can occur at any age but is most commonly seen during 40 – 60 years of age and is unusual before puberty. [63]

In our study the common age group affected was between 31-60 years.

Incidence of onychomycosis was more in females than males attributed due to higher number of female respondents in our study. The sex ratio is [1:1.4].

Among the females the majority were housewives 37/52 (71%). This is due to frequent contact with water and detergent that damages the cuticle, which then favours the invasion by fungi.

Trauma was the major predisposing factor in our study, followed by diabetes mellitus. A study by Dogra S, Kumar B, Bhansali A et al., concluded that diabetics were 2.5 times more likely than controls to have onychomycosis because of impaired circulation and peripheral neuropathy.

The number of patients with each individual disease was too low to draw any conclusions on the prevalence of onychomycosis in conjunction with these individual diseases. (Vitiligo,Psoriasis,Hansen's, Hypothyroidism, Intertrigo, Keratolysis punctata, Perforating folliculitis, Renal transplant, Filaria, Morphea, Seizure disorder).

In our study finger nails were involved more often than the toe nails. This has also been reported by other authors (64, 65). A higher incidence of fingernail onychomycosis may be a result of the increased chance for trauma. Moreover, finger nail infection is more likely to arouse the patient's concern.

Distal and lateral subungual onychomycosis was the most common clinical pattern observed in our study which is in agreement with a study by Garg A, Venkatesh V, singh M et.al, in 2004.

In this study, direct microscopic examination was positive in 25.8% samples, culture yields growth in 15.7%.Both positivity was reported in 58.4% of the samples.

Although the microscopic examination is simple and gives quicker results, culture remains the gold standard because of its specificity for genus and species identification.

In our study nondermatophyte moulds were isolated in majority (51.8%) followed by yeasts (26.8%), dermatophytes (21.4%). Until recently, yeasts were regarded as contaminants but increasingly they are emerging as pathogens.[66]

The role of aspergillus species as pathogens has been a topic of controversy as they are commonly considered as contaminants. However various recent studies and case reports have confirmed its pathogenic role.

Grover reported Aspergillus niger as a causative pathogen of onychomycosis[67]. Veer et.al, also reported 50%. Aspergillus species isolated out of total 13.6% NDM fungal isolates[64].

Ramani et.al, reported curvalaria as causative pathogen in IJDVL may 2004.

Onychomycosis is known to be difficult to treat and often exerts a significant negative impact on the quality of life. The agents most commonly used for treatment of onychomycosis are Griseofulvin, oral Fluconazole, Itraconazole, Terbinafine and topical ciclopiroxolamine and amorolfine.

The newer antifungal agents have better pharmacokinetic profiles such as prompt penetration of the nail and nail bed, persistence in the nail for several months even after the discontinuation of therapy and fewer adverse reactions.

Baran et.al, studied the efficacy of a combination therapy with amorolfine nail lacquer and oral terbinafine in comparison to oral terbinafine alone for the treatment of onychomycosis and concluded combination enhances clinical efficacy and is more cost effective[68].

In our study even though the combination therapy gave mycological cure rate higher than terbinafine monotherapy it does not have statistically significant difference (p value 0.62) in efficacy than that of terbinafine monotherapy which is also cost effective and ensuring that the patients are likely to complete the therapy.

CONCLUSION

- Females are more commonly affected than males.
- Common age group is between 31-60 yrs
- Trauma, Diabetes were found to be the precipitating factor.
- Finger nail involvement is found more than toe nail involvement.
- Distal and lateral subungual onychomycosis is the commonest type.
- Nondermatophyte moulds are commonly isolated than dermatophytes , yeasts.
- No specific systemic disease association is noted in this study.
- Therapeutic trial concludes that terbinafine monotherapy is as good as combination therapy.

The clinicoetiological correlation reveals that a single pathogen may give rise to various clinical types. The recognition of the changing prevalence of etiological agents will aid in the therapeutic approach and the potential implementation of the control measures. The efficacy, short duration, lesser side effects, cost effectiveness of the drug (particularly Terbinafine) gives a positive approach in the treatment of onychomycosis.

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MASTER CHART

NAME	AGE / SEX	OCCUPATION	DURATION	TRAUMA	HT	DM	FAMILY HISTORY	SMOKING	ALCOHOLIC	FN	TN	CLINICAL TYPE	TP / TM / TC	OTHERS	LAB DIAGNOSIS	TRIAL GROUP	MYCOLOGICAL CURE	CLINICAL CURE
SUBRAMANI	47/M	Manson	4 Y	-	-	-	-	-	-	-	3	TDO	-	-	KOH +;C- Aspergillus niger	A	+	+
ANDAL	60/F	House wife	2 Y	-	-	-	-	-	-	2	10	PSO/TDO	-	-	KOH +;C- Aspergillus niger	B	-	-
JESIMA	37/F	House wife	6 M	-	-	-	-	-	-	3	5	PSO	-	KP/I ntertrigo	KOH +;C- Nonalbicans candida	C	+	+
SANTHA	42/F	House wife	2 M	-	-	-	-	-	-	3	1	DLSO/TDO	-	-	KOH +; C- Neg	-		
SARASWATHY	60/F	House wife	1 Y	-	+	-	-	-	-	4	2	DLSO	-	-	KOH +; C- Rhizopus	A	+	+
SUNDAR	42/M	Manual labourer	1 M	+	-	-	-	+	-	3	6	PSO/TDO	-	TV	KOH +; C- Aspergillus niger	B	-	-
RAMZAN	40/F	House wife	4 Y	-	-	-	-	-	-	7	5	TDO	-	-	KOH +; C- Candida albicans	C	+	+
DEVADAS	38/M	Contractor	4 Y	-	-	-	-	-	-	4	2	TDO	-	-	KOH +; C- Neg	-		
SANTHABAI	60/F	Water board worker	4 Y	-	-	-	-	-	-	7	4	TDO	-	-	KOH +; C- Nonalbicans candida	A	+	+
KABITHA BEGAM	37/F	House wife	1 Y	+	+	+	-	-	-	-	7	TDO	-	Vitiligo	KOH +; C- Aspergillus niger	B	-	-
PRASITHA	40/F	House wife	4 M	-	-	-	-	-	-	10	5	DLSO/TDO	-	-	KOH +; C- Aspergillus niger	C	+	-
CHELLAMMAL	40/F	House wife	3 Y	-	-	-	-	-	-	6	2	DLSO/TDO	-	-	KOH -; C- Aspergillus niger	-		
DHANALAKSHMI	26/F	House wife	5 Y	-	-	-	-	-	-	3	2	PSO	-	-	KOH+; C- Curvalaria	A	-	-
GOPI	29/M	Teacher	10 Y	-	-	-	-	-	-	1	-	DLSO	-	-	KOH +; C- Aspergillus niger	B	-	-
NARESH	15/M	Student	1 Y	-	-	-	-	-	-	4	-	TDO	-	-	KOH +; C- Syncephalastrum	C	-	-
MEENACHI	28/F	House wife	6 M	-	-	-	-	-	-	5	-	DLSO	-	-	KOH -; C- Rhizopus	-		
PARVATHY	60/F	House wife	3 Y	-	+	-	-	-	-	-	3	TDO	-	-	KOH +; C- Neg	-		
DHAVAMANI	58/F	Water board worker	6 M	-	-	-	-	-	-	5	10	TDO	+	-	KOH +; C- Aspergillus niger	A	+	+
GANESAN	50/M	Barber	6 M	-	+	-	-	+	-	7	-	TDO	-	Hansen`s	KOH +; C- Trichophyton rubrum	B	-	-
VELLAAISAMY	65/M	Cloth seller	6 M	-	-	-	-	+	+	7	8	TDO	-	-	KOH +; C- Rhizopus	C	+	+

KASTHURI	52/F	House wife	6 M	-	-	-	+	-	-	-	2	DLSO	+	Hypothyroidism/Vitiligo	KOH -; C- Aspergillus niger	-			
GANDHIMATHI	57/F	House wife	2 M	-	-	-	-	-	-	-	2	DLSO	+	-	KOH +; C- Neg	-			
SRIDHAR	18/M	Student	1 Y	+	-	-	-	-	-	1	-	DLSO	-	-	KOH +; C- Neg	A	+	+	
VALLI	17/F	Bottle washer	1 Y	-	-	-	-	-	-	-	1	DLSO	-	-	KOH +; C- Candida albicans	B	+	+	
VACHALLA	36/F	Office staff	2 Y	-	-	-	+	-	-	1	-	DLSO	+	Pitting	KOH +; C- Candida albicans	C	-	-	
SUNDARI	38/F	House wife	4 Y	-	-	-	-	-		2	2	DLSO	-	-	KOH -; C- Aspergillus niger	-			
PARIMALA	44/F	House wife	3 M	-	-	-	-	-	-	2	-	DLSO/TDO	-	-	KOH -; C- Aspergillus niger	-			
MAHESH	20/F	Sweeper	6 M	+	-	-	-	-	+	5	-	TDO	-	-	KOH +; C- Curvalaria	A	+	-	
VALLIAMMAL	70/F	House wife	1 Y	+	-	+	-	-	-	-	5	TDO	-	TV	KOH +; C- Nonalbicans Candida	B	-	-	
RENUGA	32/F	House wife	3 M	-	-	-	-	-	-	5	2	DLSO	-	-	KOH +; C- Nonalbicans candida	C	+	+	
PARVATHY	50/F	House wife	1 Y	-	-	-	-	-	-	4	2	TDO	-	Pitting	KOH -; C- Aspergillus niger	-			
MARIMUTHU	32/M	Office staff	2 M	-	-	+	-	-	-	7	6	TDO	-	Perforating folliculitis	KOH +; C- Neg	-			
RAJAN	21/M	Postal staff	1 Y	-	-	-	-	-	+	2	-	DLSO/TDO	-	-	KOH +; C- Syncephalastrum	A	-	-	
SARASWATHY	59/F	Juice maker	1 Y	-	-	+	-	-	-	3	2	DLSO	-	-	KOH +; C- Aspergillus niger	B	-	-	
KANNAN	54/M	Office staff	2 Y	-	-	-	-	-	-	6	4	PSO/TDO	-	-	KOH +; C- Trichophyton rubrum	C	+	+	
RAMATHAI	43/F	House wife	3 M	-	-	-	-	-	-	4	2	DLSO/TDO	-	-	KOH +; C- Neg	-			
MAHESHVARI	35/F	House wife	8 M	-	-	-	-		-	3	-	TDO	-	-	KOH +; C- Neg	-			
MALLIGA BEEVI	65/F	House wife	3 M	-	+	-	-	-	-	-	6	DLSO	-	-	KOH +; C- Trichophyton verrucosum	A	+	+	
BHAVANI	26/F	House wife	2 Y	-	-	-	-	-	-	4	-	DLSO	-	Morphea	KOH +; C- Aspergillus niger	B	+	-	
LAKSHMI	38/F	Rice mill labourer	4 Y	-	-	-	-	-	-	6	3	DLSO/TDO	-	-	KOH +; C- Trichophyton schoenlenii	C	+	+	
RAMTHULASI	60/F	Farmer	10 Y	-	-	-	-	-	-	3	3	TDO	-	-	KOH -; C- Aspergillus niger	-			
KALIYAN	45/M	Farmer	8 M	-	-	-	-	-	-	2	10	DLSO	-	Hansen`s	KOH +; C- Neg	-			
LATHA	25/F	House wife	1 Y	-	-	-	-	-	-	1	-	DLSO	-	-	KOH +; C- Neg	-			
SRIPAL	59/M	Driver	2 M	-	-	-	-	+	+	5	-	PSO	-	Psoriasis	KOH +; C- Aspergillus niger	A	-	-	

MANIKANDAN	18/M	Student	3 Y	-	-	-	-	-	-	-	2	DLSO	-	-	KOH +; C- Rhizopus	B	+	+
ANJALIDEVI	52/F	House wife	3 M	-	+	+	-	-	-	1	-	PSO	-	-	KOH -; C- Trichophyton rubrum	C	+	+
SORNAKILI	60/F	House wife	2 M	-	-	+	-	-	-	2	-	TDO	-	-	KOH +; C- Neg	-		
RAJAVALLI	55/F	Servant maid	2 M	-	-	-	-	-	-	4	1	TDO	-	Intertrigo	KOH +; C- Neg	-		
ASAN PATHIMA	3/F	-	7 M	-	-	-	-	-	-	2	-	TDO	-	-	KOH +; C- Neg	-		
SHAKILA	21/F	House wife	1 Y	-	-	-	-	-	-	10	-	DLSO	-	-	KOH +; C- Nonalbicans candida	A	+	-
SUTHAN	22/M	Student	2 M	-	-	-	-	-	-	10	-	SWO	-	Pitting	KOH +; C- Trichophyton.rubrum	B	-	-
RAVI	32/M	Welder	1 Y	+	-	+	-	+	+	2	-	DLSO	-	-	KOH +; C- Candida albicans	C	+	-
KALISHA	32/M	Chemist	3 M	-	-	-	-	-	-	4	6	DLSO	-	-	KOH +; C- Trichophyton mentagrophytes	A	+	+
KALA	55/F	Farmer	4 M	-	+	+	-	-	-	2	10	TDO	-	-	KOH -; C- Aspergillus niger	-		
NAMASIVAYAM	64/M	Watchman	5 Y	+	-	-	-	-	-	7	2	DLSO	-	Filaria	KOH -; C- Aspergillus niger	-		
SUNDARAM	30/M	Medical staff	7 M	-	-	-	-	-	-	-	1	DLSO	-	-	KOH +; C- Trichophyton schoenlenii	B	+	+
RAMBHAI	60/F	House wife	4 M	-	-	-	-	-	-	4	1	DLSO	+	-	KOH +; C- Syncephalastrum	C	+	+
PONURANGAM	47/M	Farmer	3 Y	-	-	-	-	+	-	8	2	DLSO	-	-	KOH +; C- Aureobasidium pullalens	A	-	-
RAMESH	27/M	Hotel staff	1 M	-	-	-	-	+	+	3	-	DLSO	-	-	KOH +; C- Candida albicans	B	+	+
SAROJA	65/F	Servant maid	1 M	-	-	-	-	-	-	1	-	PSO	-	-	KOH +; C- Aspergillus niger	C	+	+
NAGARATHINAM	50/F	Farmer	1 Y	-	+	+	-	-	-	2	4	TDO	-	Psoriasis	KOH +; C- Nonalbicans candida	A	+	+
GUNASEKARAN	44/M	Farmer	2 Y	-	+	-	-	-	-	6	2	TDO	-	Seizure disorer	KOH +; C- Trichophyton verrucosum	B	+	+
MURUGAIYA	58/M	Merchant	1 Y	-	-	-	-	-	-	5	2	DLSO	-	-	KOH +; C- Neg	C	+	+
ARUMUGAM	67/M	Hotel staff	6 M	-	-	-	-	+	-	5	-	DLSO	+	-	KOH +; C- Aspergillus niger	A	-	-
GOWTHAMI	55/F	House wife	3 M	-	-	-	-	-	-	3	2	DLSO	-	Vitiligo	KOH -; C- Aspergillus niger	-		
DHARA	41/F	House wife	1 M	-	-	-	-	-	-	2	4	DLSO	+	Keratolysis punctata	KOH -; C- Nonalbicans candida	-		

ALAGESAN	64/M	Manson	1 M	-	-	-	-	-	-	5	2	TDO	-	Psoriasis	KOH +; C- Rhizopus	B	+	+
VICKEY	32/M	Electrician	5 Y	+	-	-	-	+	-	1	5	DLSO	+	-	KOH +; C- Neg	C	+	+
JASWANTH	31/M	Merchant	5 Y	-	-	-	-	-	-	10	-	SWO/TDO	-	Pitting	KOH +; C- Rhizopus	A	-	-
ROSI	60/F	Office staff	5 M	-	-	-	-	-	-	-	2	DLSO	-	-	KOH +; C- Scopulariopsis	B	-	-
SUSIRAJ	42/M	Driver	1 Y	-	-	+	-	-	-	2	-	DLSO	+	-	KOH +; C- Neg	C	+	+
RAMESH	21/M	Office staff	2 M	-	-	-	-	-	-	3	-	DLSO	+	Renal transplanted	KOH +; C- Candida albicans	A	-	-
REVATHI	18/M	Student	5 Y	-	-	-	-	-	-	3	-	TDO	-	Nevus achromicus	KOH +; C- Trichophyton.violaceum	B	+	+
RUKUMANI	35/F	House wife	3 M	-	-	-	+	-	-	5	2	DLSO/TDO	-	-	KOH +; C- Syncephalastrum	C	+	+
GLARA	50/F	House wife	3 M	-	-	-	-	-	-	1	-	PSO	-	-	KOH +; C- Aspergillus niger	A	+	+
RAMANI	49/F	House wife	1 Y	-	-	-	-	-	-	2	5	DLSO	-	-	KOH +; C- Aureobasidium pullalens	B	+	+
GOPALAKRISHNAN	45/M	Welder	2 Y	+	-	+	-	+	+	5	5	DLSO/TDO	+	-	KOH +; C- Aspergillus niger	C	+	+
PRABAKARAN	30/M	LIC agent	1 M	+	-	-	-	-	-	1	-	DLSO	-	-	KOH -; C- Trichophyton.rubrum	A	+	+
PECHIAMMAL	59/F	House wife	2 Y	+	-	-	-	-	-	1	-	DLSO	-	-	KOH +; C- Candida albicans	B	-	-
MOHAIDEEN	40/M	Electrician	6 M	-	-	-	-	+	+	4	2	PSO/TDO	-	Onychomadesis	KOH +; C- Neg	-		
RANCHITHKUMAR	30/M	Businessman	1 M	-	-	-	-	-	-	2	-	DLSO	-	Renal transplanted	KOH +; C- Neg			
RAJENDREN	41/M	Hotel staff	3 Y	-	-	-	-	+	+	8	-	DLSO/TDO	-	-	KOH +; C- Neg	C	-	-
MURALI	17/M	Student	1 M	-	-	-	-	-	-	1	-	PSO	+	-	KOH +; C- Neg	-		
ANITHA	29/F	House wife	3 Y	-	-	-	-	-	-	2	1	TDO	-	-	KOH -; C- Trichophyton mentagrophytes	A	+	+
RAJALAKSHMI	26/F	House wife	1 Y	-	-	-	-	-	-	7	3	DLSO	+	-	KOH +; C- Nonalbicans candida	B	-	-
BAMA	20/F	House wife	3 M	-	-	-	-	-	-	1	-	DLSO	-	-	KOH +; C- Neg	-		
INDHUMATHI	18/F	Student	4 Y	+	-	-	+	-	-	5	-	DLSO	-	-	KOH +; C- Neg	-		
SUMATHI	38/F	House wife	2 Wks	-	+	-	-	-	-	5	-	TDO	+	-	KOH +; C- Aspergillus niger	C	-	-
PANDURENGAN	54/M	Medical staff	6 M	-	-	-	-	-	-	2	-	DLSO	-	-	KOH +; C- Neg	-		

KEY TO MASTER CHART

Sex

M - Male
F - Female

Duration

Y - Years
M - Months
Wks - Weeks

Past H/o

HT - Hypertension
DM - Diabetes mellitus

Nail

FN - Finger Nail
TN - Toe Nail

Clinical types

DLSO –Distal and lateral subungual onychomycosis PSO – Proximal subungual onychomycosis
SWO – Superficial white onychomycosis
TDO – Total dystrophic onychomycosis

Associated Dermatological Condition

TP - Tines pedis
TM - Tinea manum
TC - Tinea corporis

Other Association

KP - Keratolysis punctata
TV - Tinea versicolar

Lab Diagnosis

KOH - Potassium Hydroxide Examination

C - Culture

Neg - Negative

\+ - Positive

\- - Negative

PROFORMA

Serial Number

Name Date

Age Address

Sex

Occupation

Complaints:

Duration

Onset

Site

Associated symptoms

Precipitating factor

H/o exposure to STD

Past H/o : HT/DM/Peripheral vascular Disease/ Varicose Veins /

Immunosuppressive therapy/ Atopic dermatitis.

Family H/o : Other members affected or not.

Personal H/o: Smoking/Alcoholic

Vegetarian/Mixed diet.

Treatment H/o: Topical

Allopathic ↗ ↘

Systemic

Topical

Indigenous ↗ ↘

Systemic

General Examination:

Anaemia / Pedal edema / Jaundice / Clubbing/Cyanosis /

Generalized lymphadenopathy

CVS PR

RS BP

Abdomen

CNS

Musculoskeletal System

Nail:

Toe nail / Finger nail

Number of nails

affected :

Site of origin :

Discolouration : Yellow / Brown / Black

Thickening / Ridging

Subungal hyperkeratosis

Onycholysis

Dystrophy

Paronychia

Types : DLSO / PSO / SWO / TDO /

T.manum / T.pedis

Hair :

Mucosa :

Skin :

Others :

LAB INVESTIGATION

Blood

Hb%	SGOT
TC	SGPT
DC	SAP
ESR	STP
Platelet Count	STB
B.Sugar	KOH Mount
B.Urea	Culture
S.Creatine	Biopsy

Urine

Albumin

Sugar

Deposits

RX GIVEN

Topical Systemic

Response to therapy

Follow up

Remarks